CLINICAL MEDICINE
FOR
NURSES

PAUL H. RINGER, M.D.

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BY

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ILLUSTRATED

TO THE NURSES OF THE
ASHEVILLE MISSION HOSPITAL, ASHEVILLE, N. C.,
WHOSE NEED HAS BEEN THE MAIN
STIMULUS TO THE WRITING
OF THIS VOLUME.

PREFACE.

THE following chapters represent the substance of lectures on medical diseases that I have delivered for several years at the Asheville Mission Hospital.

I have been impelled to write them out, as in no textbook for nurses that I have seen did I feel that the subjects were taken up in sufficient detail, while in all textbooks on medicine there were far too many minutiæ for the pupil nurse to attempt to master.

The object of these lectures is to place in concrete form a fairly detailed description of the points in the various diseases that nurses will be expected to observe and interpret, and also to form a basis upon which class-room lessons can be assigned and quizzes held, the teacher amplifying as he sees fit.

It will be noted that the bacteriology and pathology of diseases, save in a very few instances, have been but sketchily traced. The main points dwelt upon have been symptoms and their meaning, complications and their detection, as far as the nurse is concerned. Physical signs have been wholly set aside. I do not feel that any nurse's mind should be burdened with their description and significance.

Treatment has been dealt with in a general manner, it being ever borne in mind that each physician has his own preference for the treatment of almost every disease, and that such preference should not be infringed upon in a textbook intended solely for teaching purposes by physicians with varied ideas. I do not believe that any of the principles set forth will seriously offend.

The subject matter of the lectures has no claim whatsoever to originality. I have freely consulted the best authorities at my command, abstracting here and there in order to produce a concrete whole. The only claim that these lectures have for individuality lies in their being, as far as I know, the first collection of lectures covering a considerable number of medical diseases delivered for and to nurses.

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CHAPTER I.

FEVER.*

PROBABLY the most common single symptom of acute disease is fever. It would be unwise and undesirable to go at any length into the origin of fever, for it is veiled in much mystery and in many theoretical considerations; suffice it to say that it occurs in various degrees of intensity in a host of maladies, that it is one of the best signs we have of disturbance in the human body, and that it is the one symptom above all others that by its presence speaks for the existence of some pathological condition, though giving usually no clue to the location of the trouble.

Fever, itself, while called a "symptom," is really a "symptom-complex," being a very complete clinical entity, quite irrespective of the underlying cause. The ordinary symptoms of fever of moderate severity (*e.g.*, 103°) are:

Hot, dry skin.
Flushed face.
Bright, anxious eyes.
Thirst.
Full, rapid and bounding pulse.
Rapid and rather shallow respiration.
Headache.
Anorexia.
General aching in body and limbs.

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* Throughout this book the Fahrenheit scale is used, unless otherwise specified.

Nausea and vomiting (not very frequently).

Constipation or diarrhea (usually the former).

Scant, high-colored urine.

Of course, at times some of these symptoms will be more intense, and at times others, but in the main all of them will be present to some extent. When the temperature rises to 105° or over, or even below this limit in susceptible individuals, delirium may occur. This may be accompanied by convulsions, especially in children. Should the patient be extremely toxic, coma may set in —always a very serious sign.

Fever may be :

1. Continuous, *e.g.*, scarlet fever.

2. Remittent, a fever that shows rises and falls, but that never wholly leaves the patient, *e.g.*, typhoid fever, septic fevers.

3. Intermittent, a fever that seizes the patient, and then wholly disappears, only to return again, *e.g.*, the fever in tertian and quartan malaria.

Fever may begin :

1. Suddenly, *e.g.*, lobar pneumonia.

2. Gradually, *e.g.*, typhoid fever.

Fever may end :

1. By crisis, *i.e.*, suddenly ; *e.g.*, lobar pneumonia.

2. By lysis, *i.e.*, gradually ; *e.g.*, typhoid fever, measles.

Fever is divided into the following classes by Wunderlich :

1. Subfebrile, 99.5° to 100.4°.

2. Slightly febrile, 100.4° to 101.3°.

3. Moderately febrile, 101.3° to 103.1°.

4. Decidedly febrile, 103.1° to 104°.

5. Highly febrile, above 103.1° in the morning and
above 104.9° in the evening.

6. Hyperpyretic, above 106°.

There is usually a certain ratio between the height of
the temperature and the pulse-rate. Thus, generally
speaking

A temperature of   98.4 corresponds to a pulse-rate of   70
"   "   " 100.   "   "   "   "   "   80- 90
"   "   " 102.   "   "   "   "   "   100-110
"   "   " 104.   "   "   "   "   "   120-130

These rules are capable of the utmost variation.

CHAPTER II.

FOOD AND NUTRITION.

ALL food may be regarded as fuel, and the body as the furnace in which it is consumed. The object of food is to supply nutriment to the body, and this nutriment is used in the production of heat and energy. When taken into the body, food undergoes the following processes:

1. Digestion.
2. Absorption.
3. Oxidation.
4. Excretion.

There are five main classes of foodstuffs:

1. Proteins.
2. Fats.
3. Carbohydrates.
4. Mineral salts of

(w) Calcium
(x) Sodium
(y) Potassium
(z) Magnesium
} 6 per cent. of body weight.

5. Water—60 per cent. of food, and serving, among other things, to keep the body at a proper consistency.

1. *Proteins.* These include all foodstuffs containing nitrogen (N), and are absolutely indispensable to the maintenance of life. An animal fed on a protein-free diet, no matter how generous or how abundant it may be, eventually starves to death.

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Proteins are divided into several sub-classes, but three of which will be mentioned:

    I. Albuminoids.
        (a) White of egg.
        (b) Curd of milk.
        (c) Lean of meat.
        (d) Gluten of wheat.
    II. Gelatinoids.
        Gelatin the best example.
    III. Extractives.
        Contain nitrogen, but differ from the two preceding in that they merely add zest to the food, but have practically no nutritive value.

2. *Fats.* Fats, roughly speaking, make up about 15 per cent. of the average individual, and are generally taken into the body in the form of:

        Butter.
        Cream.
        Oils.

The amount of fat varies very greatly in different individuals.

3. *Carbohydrates.* These substances are formed of carbon (C), hydrogen (H) and oxygen (O), the last two always appearing in the proportion to form water—H_2O. Thus, starch has the chemical formula $C_6H_{10}O_5$. It will be noted that the atoms of H are just twice as numerous as those of O; thus we have the proportion H_2O. Bread, rice, and potato are examples of carbohydrate foods.

OBJECT OF VARIOUS FOODSTUFFS.

1. *Proteins.* These are to build up tissue, and to a certain extent to be converted into other foodstuffs, such as

fat and carbohydrate. They also serve as fuel to yield heat and muscular power.

2. *Fats*. These form an abundant source of heat and energy. They are also useful in serving as a buffer to the body at various points where much friction occurs, and in addition form the main reserve and storehouse upon which the body can make demands in times of necessity. Thus, when food is withheld, the body needs are primarily supplied by the overplus of fat. The emaciation consequent upon long illness is chiefly due to the oxidation and using up of the reserve store of fat.

3. *Carbohydrates*. In addition to their intrinsic food value, these substances may be transformed into fats, and used as such, or else they may be used as fuel to supply the immediate body needs.

As mentioned above, *protein* is the substance without which life cannot be sustained. Protein can, to a certain extent, be transformed into fat and carbohydrate, and, as seen above, carbohydrate can be transformed into fat; but neither fat nor carbohydrate can in any way be transformed into protein, for neither of them contains the all-important element, nitrogen. Hence, a protein-free diet amounts in the long run to starvation, and, if persisted in for a sufficient length of time, proves fatal.

From the foregoing it must not be imagined that a fat-free or a carbohydrate-free diet would in any way prove nourishing, beneficial or healthful. All three classes of foodstuffs are of paramount importance to the human body, and must be taken in certain well-defined general proportions; but stress must be laid on the fact that *protein* is an absolute necessity.

The energy and heat of the body are derived from the combustion of its foodstuffs. As energy can be con-

verted into heat, the value of foodstuffs can be expressed in heat units. The heat unit is called *calorie*. A calorie is that amount of heat needed to raise a kilogram of water one degree Centigrade.

The amount of heat given off from the human body has been measured with accuracy. In a condition of rest, a man gives off heat in twenty-four hours equivalent to about 33 calories per kilogram (2.2 pounds) of body weight. Thus, a man weighing 70 kilos (154 pounds) gives off about 2310 calories (70 × 33). This amount must be supplied by foodstuffs in order to maintain a satisfactory state of nutrition. It has been calculated that these 2310 calories must contain about 500 Gm. of carbohydrate, 50 to 100 Gm. of fat, and 120 Gm. of protein.

Rubner's figures are usually taken in calculating the caloric value of foods. According to his figures:

1 Gm. of protein produces 4 calories.

1 Gm. of fat produces 9.3 calories.

1 Gm. of carbohydrate produces 4.1 calories.

Rubner gives the following standard dietary for a man of 70 kilos (154 lbs.):

	Light work	Medium work	Heavy work
Protein Gm.	123	127	165
Fat Gm.	46	52	70
Carbohydrate Gm.	377	509	565
Calories	2445	2868	3362

Thus it will be seen that the average man doing medium work requires about 127 Gm. of protein daily. Not much more than 150 Gm. can be given without the appearance of symptoms of overfeeding—gastric or intestinal derangements. In feeding any individual, the point

to be borne in mind is not so much the maximum or
minimum number of calories which the patient will toler-
ate, or upon which he can exist, as that amount upon
which the patient will thrive best.

The following table gives the daily needs in calories
of an adult weighing 65 kilos (162.5 lbs.) :

1. During rest in bed 1800 cal., or 28 cal. per kilo of body wt.
2. In repose   2100 "   " 32 "   "   "   "   "   "
3. Light work   2300 "   " 33 "   "   "   "   "   "
4. Moderate work   2600 "   " 40 "   "   "   "   "   "
5. Hard work   3100 "   " 48 "   "   "   "   "   "

Infants require more calories per kilo of body weight
than do adults. This can readily be accounted for when
we consider the tremendous growth and consequent tis-
sue changes taking place in the infant.

For 1st 3 mos. an infant requires 100 cal. per kilo of body wt.
 " 2d 3 "   "   "   " 90-100 "   "   "   "   "   "
 " 2d 6 "   "   "   "   80 "   "   "   "   "   "

Average cow's milk contains 320 calories per pint (640
calories per quart). Eggs contain about 720 calories per
pound, the whites alone yielding 250 calories per pound,
and the yolks 1705 calories per pound. The white is pure
protein, while the yolk contains numerous substances,
chief of which are: 15 per cent. protein, 20 per cent.
fat, besides lecithin, nuclein, salts of iron, calcium,
potassium, and magnesium.

Meats are best prepared by broiling or roasting. Bouil-
lons and beef extracts consist mainly of extractives from
the meat, and, contrary to an idea almost universally
prevalent among the laity, have practically no food value.
The following table gives the caloric value per pound
of the principal meats:

Calories

Beef (steak) 975
Veal 745
Mutton 890
Lamb 1075
Pork chops 1245
Chicken (broilers) 305
Turkey 1060

Vegetables contain a large percentage of starch and sugar, and a somewhat lesser percentage of protein.

The number of calories needed daily by a man in health has been dwelt upon in some detail. When an individual is suffering with fever from any cause, from 20 to 30 per cent. more heat is given off than in health. This must be made good by an increased caloric intake, or the patient will suffer. Especially is this true in long fevers, such as those caused by typhoid, tuberculosis, and rheumatic fever. In the shorter fevers, such as lobar pneumonia, the maintenance of the bodily strength by means of increased caloric feeding is not so important. If 25 per cent. be added to the normal amount required by the average man (2300 calories in 24 hours), we see that during fever from 2800 to 2900 calories in 24 hours will be needed.

Foods whose caloric value is not very great can have that value raised by the addition of substances whose caloric value is very high, such as milk sugar (caloric value per ounce, 117) and cream (caloric value per ounce, 54). Qualitative changes can be made in foods which will counteract the enormous quantity that would have to be ingested to supply the caloric needs were the food, as such, given. For instance, if in a case of typhoid reliance were placed solely on a milk diet, and the stock order, "a glass of milk every two hours" carried into

effect, the patient would be wretchedly under-nourished. A glass of milk contains from 6 to 8 ounces. One quart of milk produces 640 calories. Ten feedings will be about all the patient will get in 24 hours. He will, therefore, be given from 60 to 80 ounces of milk—practically two quarts, 1280 calories—two-thirds of what he should really have. To meet the needs of the patient, five quarts of milk daily would be required—an obvious absurdity and impossibility. Latterly, the so-called "high-calorie diet" has been used with marked success in the treatment of typhoid fever, further reference to which will be made in the chapter on that disease.

Administration of Food to the Sick.

Details—one might almost say trivialities—are of the greatest importance, and are too often not sufficiently heeded by the nurse. Florence Nightingale wrote: "To watch for the opinions which the patient's stomach gives rather than to read 'analyses of foods' is the business of all those who have to settle what the patient is to eat— perhaps the most important thing to be provided for him after the air he breathes.   *   *   *   An almost universal error among nurses is the bulk of the food, and especially of the drinks, they offer to their patients. It requires very nice observation and care (and meets with hardly any) to determine what will not be too thick or too strong for the patient to take, while giving him no more than he is able to swallow."

The following are some important points to be noted in the feeding of the sick:

1. *Punctuality*. To the invalid meal-time is an important event. He looks forward to it with interest and with

curiosity. He eyes the clock a hundred times until the arrival of the appointed hour. When that hour comes, the meal also should come. Waiting until the stated time tends to sharpen the patient's appetite; waiting beyond that time disappoints, irritates, and tends markedly to blunt the desire for food.

2. Do not ask the patient with a poor appetite what he wants to eat. He does not want anything, and if foods are named to him and his suggestions invited, his repugnance becomes increased. His appetite can best be stimulated by exciting his surprise and curiosity.

3. Untasted food, dishes after use, half-emptied cups and glasses, should never be left in the sick-room. They are unsanitary, and often tend to nauseate the sensitive patient. There is nothing more frequently seen in the sick-room, and there are few things more disgusting, than an empty, unwashed glass that has contained milk.

4. Wipe dishes dry on the *outside*, and take special care that the contents of cups are not spilled into the saucers.

5. Mutton or chicken broth should be skimmed several times before serving. Blotting paper or a piece of thread can be passed over the surface to remove the last traces of oily substance.

6. When the dietary is limited, or the appetite is poor, it is often well to serve the meal in "courses." Time after time the writer has had patients complain that they lost all desire for food at once after the appearance of a large tray loaded down with all sorts of eatables, from soup to dessert. Many of these same patients would have eaten well and with enjoyment had the same food been daintily served, one course at a time.

7. Do not offer food to the patient immediately after a bowel or bladder evacuation. If the patient has just used the bed-pan or urinal, the nurse should make it very apparent that she has thoroughly cleansed her hands before busying herself with food.

CHAPTER III.

THE CIRCULATION—GENERAL CONSIDERATIONS.

THE vascular system in and through which the blood circulates consists of a central pump (the heart) and a system of tubes of three distinct types:

1. *Arteries.* Vessels carrying blood *away from* the heart, becoming progressively *smaller* as their distance from the heart increases, having walls relatively thick, rich in elastic fibres, by the recoil of which the blood stream generated by the force of the heart-beat is kept in motion, and in which the blood is kept at a relatively high degree of pressure.

2. *Capillaries.* Microscopic vessels everywhere permeating the tissues, lined by a single layer of cells, through the walls of which the food and oxygen brought by the blood are taken up by the tissues, and the waste products to be gotten rid of by the organs of elimination are given off.

3. *Veins.* Relatively large vessels, in comparison with the arteries, becoming progressively *larger* as they approach the heart, bearing stale and deoxygenated blood *toward* the heart, having thin walls, poor in elastic fibres and easily collapsible, containing the blood under very low pressure, and possessing at frequent intervals small valves to prevent any appreciable back-flow of blood.

The circulation—regular, incessant, and rhythmic—of the blood in this closed system of tubes was first discovered and demonstrated by William Harvey in 1616. In order to better understand the meaning and causation

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of symptoms in many diseases to be dealt with later, a glance into the realm of the physiology and mechanics of the circulation is desirable.

Course of the Circulation.
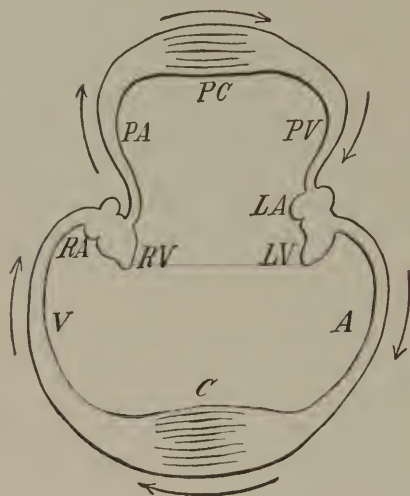
Left ventricle.

Aorta.

Fig. 1.—General diagram of the circulation: the arrows indicate the course of the blood; *PA*, pulmonary artery; *PC*, pulmonary capillaries; *PV*, pulmonary veins; *LA*, left auricle; *LV*, left ventricle; *A*, systemic arteries; *C*, systemic capillaries; *V*, systemic veins; *RA*, right auricle; *RV*, right ventricle. (From "Howell's Physiology," W. B. Saunders Co.)

Systemic arteries.

Systemic capillaries.

Systemic veins (some blood deflected through portal system; *vide infra*).

Inferior and superior venæ cavæ.

Right auricle.

Right ventricle.

Pulmonary artery (containing venous blood), named "artery" because it carries blood *away from* the heart.

Pulmonary capillaries (lungs).

Pulmonary veins (4) (containing arterial blood), named "veins" because they bring blood *toward* the heart.
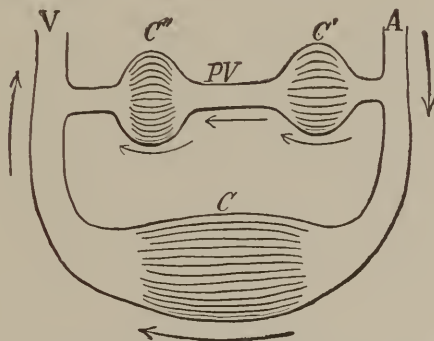
Fig. 2.—Diagram of the portal system: the arrows indicate the course of the blood; *A,* arterial system; *V,* venous system; *C,* capillaries of the spleen, pancreas, and alimentary canal; *PV,* portal vein; *C″,* capillaries of the liver; *C′,* the rest of the systemic capillaries. The hepatic artery is not represented. (From "Howell's Physiology," W. B. Saunders Co.)

Left auricle.

Left ventricle.

Subdivisions of the Circulation.

1. *Greater or systemic.* Includes all arteries, capillaries and veins throughout the body, *except* those going to and from the lungs and those vessels uniting to form the portal system (*q.v.*).

2. *Lesser or pulmonary.* Includes the pulmonary artery, the pulmonary capillaries and the four pulmonary veins—in other words, all those vessels in which the blood is contained from the time it leaves the right ventricle until it enters the left auricle.

3. *Portal or splanchnic.* Includes those veins (and veins only) which drain the alimentary tract, and whose blood, consequently, is more filled with waste products than is that from other portions of the body. These veins—the so-called "radicals"—of the portal system join to form the *portal vein* which, in company with the hepatic artery and the common bile duct, enters the liver embedded in the capsule of Glisson, and in the liver breaks up into branches, and delivers its blood to that organ to be further purified before leaving through the hepatic vein to proceed onward toward the heart.

The physiology of the circulation, especially that of the lesser or pulmonary circulation, is so intimately blended with the fundamentals of the physiology of respiration that a few lines on that subject will not be amiss.

Respiration and the lungs provide for an exchange of gases between the blood on the one hand and the external air on the other. The blood coming to the pulmonary capillaries from the right side of the heart via the pulmonary artery, is blood that has made its rounds of the circulation, has given up to the tissues the oxygen that it had acquired at its previous passage through the lungs, and has taken unto itself instead waste products absorbed from the tissues during its passage through them. It must now yield up its waste products, and take unto itself a new supply of oxygen. This can be done in the pulmonary capillaries. These microscopical vessels have walls of extreme thinness, and through the single layer

of endothelial cells forming these walls the interchange of gases takes place during the few seconds that any given red blood corpuscle is flowing through the lungs.

The blood corpuscle gives off carbon dioxide (CO_2), and in exchange receives a full charge of oxygen from the air inspired during its sojourn in the lungs. Refreshed and ready for its duty, the corpuscle is then carried by the pulmonary veins to the left side of the heart, whence it is started on its trip over the body to give up in turn to the needy tissues the oxygen without which they cannot live.

The air in the alveoli or terminal chambers of the lungs is kept fresh and ever changing by the respiratory movements, pure air, rich in oxygen being inspired, and stale air being expired.

The Heart. The heart, or central pump, power-house and motive force of the entire circulatory system, demands notice. It is composed of muscle, striated, but involuntary—that is to say, possessing the striæ common to voluntary muscles, but being quite beyond the power of the will, though most susceptible to the emotions.

The heart contains four cavities or chambers. The two upper and smaller ones are the auricles, right and left, respectively. The two lower and larger ones are the ventricles, also right and left, respectively. The right auricle opens directly into the right ventricle, and the left auricle into the left ventricle. The superior and inferior venæ cavæ open into the right auricle. The pulmonary artery opens out from the right ventricle. The four pulmonary veins, two from each lung, open into the left auricle. The left auricle empties directly into the left ventricle; and the aorta, the main arterial trunk of the body, emerges from the upper part of that chamber.

The Cardiac Cycle. From the beginning of one beat of the heart to the beginning of the next succeeding beat constitutes a "cardiac cycle." The cardiac cycle is divided into two main parts:

1. *Systole*—the *work* portion of the cycle—that portion during which the auricles and ventricles contract and drive the blood onward.

2. *Diastole*—the *rest* portion of the cycle—that portion during which the cavities of the heart are being filled with blood which will be forced onward at the next systole.

The contractions of auricles and ventricles do not take place at exactly the same time, the auricles contracting immediately before the ventricles.

The ordinary complete cardiac cycle with the heart beating 72 times to the minute, lasts eight-tenths of a second. Of this:

 (*x*) Auricular systole lasts 0.1 second.
 (*y*) Ventricular systole lasts 0.3 second.
 (*z*) Diastole lasts 0.4 second.

Thus it is seen that 50 per cent. of the time is spent by the heart in resting. When we consider that from birth to death the heart never has what in the ordinary sense we construe as "rest," the importance of this recuperative portion of the cardiac cycle is at once apparent.

The vagus nerve exercises an important inhibitory or slowing effect upon the heart. It acts as a brake, and prevents the heart from running away with itself. Thus, it is another potent factor established by Nature for ensuring the heart's obtaining enough rest.

The Cardiac Valves. With this multiplicity of cardiac chambers and of vessels entering and leaving these chambers, there exists a most simple and unique means

of preventing the back-flow of blood, and of maintaining the mechanics of the circulation. This unique means consists in the valves of the heart. The valves of the heart are four in number:

1. *Mitral or bicuspid valve,* situated between the left auricle and the left ventricle, opened automatically by the blood current during ventricular diastole, and similarly closed during ventricular systole, thus preventing regurgitation of blood into the left auricle, and causing all the blood in the ventricular cavity to be discharged through the aorta and thus to reach the body generally.

2. *Tricuspid valve,* situated between the right auricle and the right ventricle, opened automatically by the blood current during ventricular diastole, and similarly closed during ventricular systole, thus preventing regurgitation of blood into the right auricle, and causing all the blood in the ventricular cavity to be discharged through the pulmonary artery, and thus to reach the lungs.

3. *Aortic semilunar valves,* situated at the emergence of the aorta from the left ventricle, automatically closed by the reflux of blood after ventricular systole, and remaining closed during ventricular diastole, thus preventing regurgitation of blood into the ventricular cavity, which is being filled anew from the left auricle; automatically opened by the blood current during ventricular systole to allow the passage of blood into the aorta.

4. *Pulmonary semilunar valves,* situated at the emergence of the pulmonary artery from the right ventricle, automatically closed by the reflux of blood in the pulmonary artery after ventricular systole, and remaining closed during ventricular diastole, thus preventing regurgitation of blood into the ventricular cavity, which is being filled anew from the right auricle; automatically

opened by the blood current during ventricular systole to allow the passage of blood into the pulmonary artery.

All the valves have three cusps or flaps save the mitral, which has but two. The flaps on the tricuspid and mitral valves are large and somewhat fan-shaped, of rather thick, fibrous structure, and covered with a glistening membrane, the endocardium, which is continuous with that lining the ventricular cavity. These cusps are connected with the ventricular walls by fine, cord-like processes, the chordæ tendinæ, and do not move freely in the blood current.

The structure of the aortic and pulmonary semilunar valves is precisely similar. Both are composed of three flaps, each the shape of a half-moon, these three cusps meeting in the center of the lumen or calibre of the vessel when the valve is closed, and lying up against its wall when the valve is open. They are of much finer structure than are the auriculo-ventricular valves, are about the thickness of a piece of ordinary writing paper, translucent, and lined on their cardiac surfaces with a continuation of the endocardium that lines the ventricular cavity.

These few physiological and anatomical facts will enable us to better approach the clinical side of diseases and disturbances of the heart and circulatory system.

CHAPTER IV.

RHEUMATIC FEVER.

RHEUMATIC fever has all the ear-marks of an acute infectious disease caused by a specific micro-organism. Hitherto the particular germ has resisted discovery, so the fundamental causative factor is not known. It is believed, however, to be a particular form of streptococcus. Rheumatic fever is intimately associated with the various diseases of the heart, to be taken up shortly. Dr. Olchin, a celebrated English clinician, has said that in adults rheumatic fever is a disease of the joints, with heart symptoms secondary, while in children rheumatic fever is a disease of the heart, with joint symptoms secondary. In any event, the connection between rheumatic fever and pericarditis, endocarditis, and myocarditis is so close that it is the heart that must be watched first, last, and all the time.

Etiology. Rheumatic fever attacks children and young adults in preference to those of riper years. Males are in the majority of those affected, and the disease seems to have a predilection for those following certain occupations, more especially drivers, servants, bakers, sailors, and laborers. It will be noted that these callings often necessitate prolonged exposure to the elements, entail severe wettings, and in some cases, as in bakers, a sudden and marked change in temperature coupled with a damp atmosphere. These changes, as well as exposure, seem to be predisposing factors in the disease.

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Symptomatology. The onset of the disease is usually sudden. It may be preceded by a day or two of general malaise, accompanied by vague pains in the joints. Frequently it is ushered in by sore throat, and especially by *tonsillitis.* The patient may also have been a victim of chorea (St. Vitus' Dance) which, with tonsillitis, seems to bear an important, though as yet unexplained, relationship to rheumatic fever. The temperature rises rapidly, and ranges between 102° and 104°. There are the accompanying symptoms of fever, the tongue being moist, and covered with a white fur. There is loss of appetite, usually constipation, intense thirst, and scanty, highly colored urine. In the course of the disease, profuse acid sweats occur, which may have a sour odor.

The Joints. The large joints are usually the ones involved, especially the knees, ankles, elbows, shoulders and wrists. Occasionally the articulations between the vertebræ may be the seat of inflammation. The affected joints are swollen, hot, red, tense, tender and exquisitely painful. At times their sensitiveness is so great that the tread of some one walking in the room, or the pressure of the bedclothes is unbearable. The swelling, pain, and tenderness last a variable time, from a few days to two weeks in any given joint; but it is characteristic of rheumatic fever that while one joint is recovering another becomes involved, and thus the general picture of the disease may be prolonged several weeks.

There is marked anemia and a leukocytosis, varying between 15,000 and 30,000. The disease may continue over many weeks, the fever being usually continuous, but not excessively high, and depending in its duration upon the joint involvement and upon the presence or absence of complications. The fall in the temperature is gradual,

the symptoms slowly subsiding, leaving the patient much weakened and prostrated.

The picture of a fully developed case of rheumatic fever, with its temperature and attendant symptoms, its drenching and exhausting sweats, its exquisitely painful joints, almost prohibiting all movement, and making each position assumed by the patient seem more agonizing than the preceding one, forms one of the most distressing sights in medicine.

Complications. These are not great numerically, but are most important and severe. In fact, the terror of rheumatic fever lies not in the danger from the primary disease itself, but in the complications that may, and unfortunately do, occur in a large percentage of cases. It is usually not the immediate, but the remote effects of acute rheumatic fever that are dreaded.

Hyperpyrexia. A temperature from 105° to 108° ; occurs most frequently in the second week, is an indication of grave toxemia, is often accompanied by delirium and stupor, and is most common in the first attack. It is always serious, and if prolonged and unrelieved may prove fatal.

Cardiac Affections. These complications are among the most frequent and the most serious that are met with. According to Church, cardiac complications occurred in 494 out of 889 cases—over 50 per cent. The heart conditions can be grouped under one of the three following heads :

1. Endocarditis—valvular disease.
2. Pericarditis.
3. Myocarditis.

The symptoms of these particular conditions will not be detailed here, as they are fully taken up under their

respective headings. Suffice it at this time to say that any change in the patient's condition, not directly and unquestionably ascribable to the joints, should make the nurse consider the possibility of cardiac involvement and necessitates the utmost watchfulness.

Pneumonia and Pleurisy. These complications occur in about 10 per cent. of the cases, and are characterized by their particular symptoms, the marked rise in temperature, shallow respiration, dyspnea and knife-like pain in one side being suggestive of pneumonia, while the pain without the marked onset of other symptoms is rather characteristic of pleurisy.

Cerebral Complications:

1. Delirium due to

(*x*) Hyperpyrexia..

(*y*) Toxemia.

Always serious, as denoting intense infection and lowered resistance.

2. Coma. More serious than the preceding, due to intense toxemia, and being an evidence of poor or exhausted defensive forces.

3. Convulsions. Rare.

4. Chorea. This disease, as above stated, has an ill-understood connection with rheumatism, and, when occurring as a complication, does so usually in children.

Prognosis. The prognosis in rheumatic fever is always grave, and is so, not because of the disease itself (for few die as a direct result of the rheumatic attack), but because of the frequency and severity of the complications. In fact, the outlook depends mainly upon the presence or absence of complicating factors. All cases with cardiac involvement must be looked upon as very

seriously ill, and the outcome as very uncertain, as many weeks must elapse before it is possible to determine whether a cardiac inflammation will disappear completely or else become chronic. In the first case, the outlook is good; in the second, though life may be prolonged many years, a permanently damaged organ exists, cure is out of the question, and the ultimate outlook is, therefore, bad.

Treatment. The treatment of a patient suffering from an attack of rheumatic fever may be grouped under two general heads:

1. Treatment of the general infection.

2. Drug, or specific treatment of the infection.

1. (*a*) *Rest.* The patient with rheumatic fever should from the start, be confined absolutely to bed, the use of the bed-pan insisted upon, and no permission given for the patient to sit up in any way. There are three main reasons for insistence upon absolute rest:

(*x*) The body cells are the victims of an intoxication.

(*y*) Certain tissues in the body (the joints and possibly the endocardium, pericardium, and myocardium) are undergoing alterations incident to inflammation.

(*z*) The likelihood of cardiac complications is reduced by absolute rest.

(*b*) *The Bed.* If the patient perspires freely, the bed should be made with blankets instead of sheets, and the nurse should always bear in mind that, especially in a disease of this nature, where pressure and movement cause such agonizing pain, the bed should be made with more regard to the comfort of the patient than to symmetry of appearance. It is not harmful to allow cold air in the

room, but care should be taken that at such times the patient is well covered. If the bedclothes cause pain, it may be necessary to make cradles out of barrel hoops or wire.

The patient should wear a thin flannel nightgown, open down the front, and slit up the sleeves, so as to admit of easy inspection of joints and heart. Flannel is far more welcome than cotton, because it does not become cold and clammy after sweats.

(c) *Diet.* This should be fairly liberal, small amounts frequently given being more desirable than larger amounts at longer intervals, and reliance should be placed mainly in the following articles: milk, soups, cereals, custard, bread, rice. *Acids should be wholly avoided.* With decrease in temperature, eggs may be added, and in convalescence a gradual return to normal meals. The caloric system of feeding, though somewhat troublesome to carry out, is desirable, as then one can be sure that the fuel needs of the patient are being met.

2. *Drug or Specific Treatment.* It is a sad fact that hitherto in modern medicine we are all too seldom able to treat the disease that confronts us. We can, and we do, treat the patient with the disease, but we treat him symptomatically, meeting indications as they arise, knowing full well that if we maintain his strength, keep his bowels and kidneys acting freely, and relieve him from the most burdensome of his symptoms, Nature will carry on the fight against the enemy. In the case of rheumatic fever, we possess, however, a remedy which seems to attack the disease directly, and which, while not producing results as brilliant as do antitoxin in diphtheria, and quinine in malaria, nevertheless deserves a high place among our weapons for fighting disease. This drug is salicylic acid.

In order to obtain beneficial effects from this drug it must be administered in full doses. Salicylic acid itself may be given, or else its derivative, acetylsalicylic acid (aspirin), the latter being perhaps preferable, because less trying to the stomach. The dose of either drug in an adult is usually 15 to 20 grains every two hours. The exact manner in which salicylic acid acts upon the rheumatic poison is not known, but of its marked beneficial effect abundant clinical experience has given incontestable proof. The good results of the drug are seen upon all the symptoms—pain and swelling of joints, sweats, temperature, all abating under its proper use. Often salicylic acid is given credit for not producing the desired effect because the dosage has been too small.

The limit of tolerance on the part of the patient must, however, not be overstepped. The following conditions may arise denoting that the patient can take no more of the drug:

1. *Ringing in the ears.*
2. *Gastric disturbances:*
    (*a*) Nausea.
    (*b*) Vomiting.
3. Cardiac disturbances. Irregularities.
4. Respiratory disturbances:
    (*a*) Dyspnea.
    (*b*) Sighing respiration. ·
5. Cerebral symptoms:
    (*a*) Headache.
    (*b*) Delirium.
6. Renal complications. Any variation from the normal in the kidney functions.
7. Hemorrhage. From bowels, bladder or under skin.
8. Skin involvement. Various rashes.

Upon the appearance of any of these symptoms not accounted for by other evidences in the patient, the drug must be temporarily discontinued, and subsequently resumed in reduced dosage.

In addition, alkalies in the form of the citrate and acetate of potash and the bicarbonate of soda are frequently given in full dosage, a good guide being the keeping of the urinary reaction alkaline.

Symptomatic Treatment..

Pain. Rest is the best. Place pillows or bolsters under knees. Sometimes splints lightly put on will give relief.

Cold. The ice-bag often eases pain. Especially is it valuable in pericardial pain, so frequent with beginning heart involvement.

Heat is sometimes very effectual in allaying joint-pains. It is best applied in the form of hot fomentations, as follows: Two layers of flannel are soaked in very hot water, and wrung out of a wringer made of a crash towel. The flannel is applied singly to the joint. Applications are repeated three or four times, at intervals of ten or fifteen minutes. The joints are then sponged with water at a temperature of 75°, and wrapped in dry flannel or *non*-absorbent cotton.

Oil of wintergreen applied to joints on cotton is often of great benefit, though the pungent and all-pervading odor may be objectionable.

Blisters and the *actual cautery* over the joints may give great relief, and a fly-blister the size of a silver dollar over the pericardium is of great service in allaying pain in that region.

Codeine or *morphia* in the usual dosage may be, and often are, necessary.

Hyperpyrexia. Cold packs and cold sponges are indicated for temperatures of 104° or over, and should be given as in cases of typhoid fever (*q.v.*).

Convalescence. The patient recovering from an attack of rheumatic fever must be jealously guarded for a period longer than that required in convalescence from most acute infections. The reason for this is that the heart may be the seat of slight involvement which, if unwise liberties are allowed, may become more active, and give rise to an actual endocarditis. Consequently, many weeks of care and quiet are necessary, all exposure to cold and sudden changes of temperature must · be avoided, and, if possible, when convalescence is once established, it is well to have the patient seek a climate that is sufficiently mild to enable him to live comfortably out of doors for a few weeks. Return to the normal activities of life must be very gradual, first consideration being given to the heart, which has been the subject of such a severe strain.

CHAPTER V.

PERICARDITIS.

PERICARDITIS is inflammation of the pericardium, the membrane surrounding the heart. This membrane is composed of an outer coarse, fibrous layer, and an inner fine, serous layer. The pericardium surrounds the heart on all sides and, above, the beginning of the aorta and pulmonary artery. Below, the membrane is firmly attached to the diaphragm.

The inner or serous portion of the pericardium is subdivided into two layers; an outer one lying against the fibrous pericardium, and an inner one lying directly upon the heart muscle. Between these layers, which are in contact with one another, are found a few drops of fluid which act as a lubricant, as with each beat of the heart these two layers slide one upon the other. Inflammation, known as pericarditis, is practically limited to the serous portion of the pericardium, and does not involve the fibrous layer at all.

Etiology.

1. Rheumatic fever, the most common factor.
2. Lobar pneumonia.
3. Nephritis:
   (a) Acute.
   (b) Chronic.
4. Scarlet fever.
5. Other infections.

Pericarditis is almost always of infectious origin, and is brought about by germ action. Pericarditis is divided into two great classes:

(30)

1. Dry pericarditis.

2. Pericarditis with effusion.

Pathology.

Dry Pericarditis. Congestion of the outer and inner serous layers of the pericardium. Exudation of serum, fibrin, leukocytes.

Between the layers a meshwork of fibrin is formed which prevents the two surfaces of membrane from working smoothly, one against the other. This meshwork is frequently found to contain bacteria. The heart muscle, which lies next to the inner layer of the membrane, is secondarily affected, and the individual muscle fibres, when examined under the microscope, may show degenerative changes.

Pericarditis with Effusion. Instead of the formation of a meshwork of fibrin, with but a small amount of serum, in this variety of pericarditis the exudation of serum (fluid) is the main feature. The exudate is very abundant, and in extreme cases may amount to as much as a quart. The nature of the fluid given off from the surfaces of the membrane depends upon the nature of the infecting germ. It may be:

1. Serous—a clear, yellowish fluid.

2. Purulent—cloudy, or yellow, from the presence of pus.

3. Hemorrhagic—bloody.

Symptoms. Pericarditis rarely appears as a primary disease. It usually makes its appearance as a complication of some pre-existing infectious disease. Hence, because of the existence of another malady, those symptoms due particularly to pericarditis are apt to be masked. For instance, if in the course of rheumatic fever there is a rise in temperature, without the involvement of addi-

tional joints, or without the onset of delirium or other nervous symptoms, pericarditis should be thought of as a possibility. The following symptoms are those most characteristic of

A. Dry Pericarditis.

1. *Pain* in the region of the heart, or in the epigastrium. It may at times radiate to the front and sides of the chest. Pain is due to the abnormal friction between the layers of the pericardium, arising because of the presence of the fibrin meshwork. It may be sharp or dull, and is usually continuous. If the pain suddenly ceases while the temperature fails to drop, it is a sign that effusion has set in. Effusion stops the pain by mechanically separating the layers of the pericardial membrane, so that they no longer rub against each other.

2. *Cough.* May or may not be present. If present, is frequent, and of the dry, hacking variety.

3. *Pulse.* Invariably rapid, 120 to 140. Soft and compressible. Regular until the heart muscle is affected, when irregularities are apt to occur.

4. *Respiration.* Rapid and shallow.

5. *Temperature.* Usually raised moderately (100° to 102°), but in no way characteristic or to be relied upon, as it is affected by the fever arising from the primary disease, of which pericarditis is a complication.

6. *Sleep.* Disturbed because of pain and general nervous irritability.

Course of the Disease. One to several weeks. The inflammation may wholly disappear and complete recovery ensue. The inflammation may extend to the heart muscle, and give rise to myocarditis. Effusion may appear. The outlook in pericarditis is always grave, much depending upon the nature of the primary disease.

B. Pericarditis with Effusion. In the early stages, the symptoms are the same as those of the dry variety (*q.v.*). Later, the symptoms are those caused by the accumulation of fluid in the pericardial sac, causing pressure on the heart.

1. *Cessation of pain.*

2. *Fever and cough* usually persist.

3. *Face* is pale, and the expression is anxious. There may be slight cyanosis.

4. *Pulse.* Small, rapid, and of low tension. It becomes irregular when the effusion is large and has persisted for some time. This is a bad sign, as it indicates failure of the heart muscle. If, however, owing to some pre-existing heart disease, the pulse has been irregular right along, the outlook is not so grave. The pressure of the fluid on the heart hinders the entrance of blood into the heart more than it does the exit of blood from the heart (for blood coming to the heart is under very slight pressure as compared with that of blood leaving the heart); hence, there is dilatation of the small veins of the skin.

5. *Respiration.* Rapid and shallow, and in large effusions much embarrassed, both because of pressure of fluid on the heart and because of pressure on the adjacent lung, especially the left lower lobe.

6. *Insomnia.* Marked and intractable. Due to congestion of the brain by the damming back of the blood, and also to shortness of breath.

7. *The abdomen* may be distended with gas, due to congestion in the portal system of veins. There may be constipation or small, frequent, watery stools, because of the passage of serum from the greatly congested blood-vessels into the intestines.

3

Course of the Disease. Two weeks to two months. The effusion may become absorbed in from two to four weeks. More often, however, it shows a tendency to remain. Partial absorption may be followed by more effusion. The outlook is always very grave.

Treatment.

Dry Pericarditis. There is no specific method of treatment that can be used. The patient must be carefully watched, and every effort made to help Nature. Treatment that has been instituted for the primary disease, whatever that may be, will, of course, be continued. In most cases, absolute and prolonged rest in bed is indicated. Most physicians approve of counterirritation over the heart in the shape of:

1. Heat (hot water bags, hot bottles, mustard plaster, iodine).
2. Fly-blisters.
3. Cold. Often more effective than heat. Best applied by means of the ice-bag or cold-water coil.

Other treatment is symptomatic. Cough, insomnia, fever, etc., must be met and combated by the usual methods. Frequently, moderate doses of codeine or morphia are given to relieve pain and give much needed rest.

For rapid and violent heart action, the ice-bag gives the best results. Heart stimulants are usually not needed, especially at first, as the rapid heart action is due to irritation of the heart. and not to weakness of that organ. The bowels should, of course, be kept well open, and the diet should be bland and easily digestible, consisting in the main of milk, eggs, chicken, raw oysters, birds, toast, etc. It should not be bulky, and should be given frequently and in small amounts.

Pericarditis with Effusion. Two methods of treatment are available, the object of each being, of course, to get rid of the effusion:

1. *Elimination by catharsis,* the object of this method being to give such drugs as will abstract fluid from the tissues, and therefore indirectly remove the fluid that has collected in the pericardium. Saline cathartics are those most employed.

2. *Tapping.* (Paracentesis pericardiæ).

This is called for by:

    1. Marked shortness of breath.

    2. Marked cyanosis.

    3. Marked rapidity and weakness of pulse.

The location for tapping the pericardium is the fifth left intercostal space, very near the sternum, or else one inch from that bone, in order to avoid the internal mammary artery.

CHAPTER VI.

ENDOCARDITIS (ACUTE SIMPLE AND MALIGNANT).

ENDOCARDITIS is the name given to inflammation of the endocardium or lining membrane of the heart.

Cases of endocarditis can be classified as follows:

1. Simple endocarditis.
    (*a*) Acute.
    (*b*) Chronic.
2. Malignant or ulcerative endocarditis.

Etiology. In the vast majority of cases simple acute endocarditis is of bacterial origin. Malignant endocarditis is invariably caused by some micro-organism. Although the causative germs of some of the diseases most frequently giving rise to endocarditis have not as yet been discovered (rheumatic fever, scarlet fever, measles), still there is every reason to believe that these diseases are of microbic origin.

The following diseases most frequently give rise to simple endocarditis:

Rheumatic fever (by far the most important).
Tonsillitis.
Chorea (St. Vitus' dance).
Scarlet fever.
Measles.

The following diseases most frequently give rise to malignant endocarditis:

Septicemia.
Pyæmia.
    (36)

Fresh bacterial invasions.

Pneumonia.

Gonorrhea.

Pathology. The following changes take place in the endocardium, which normally is a smooth, shiny, glistening membrane:

1. Cloudiness of the membrane.

2. Thickening and some edema.

3. Laceration.

4. An eroded surface, necrotic from the the action of bacteria and their toxins, covered with a deposit of fibrin which forms a warty cauliflower-like mass, yellowish or reddish, the so-called "vegetation." This may occur anywhere on the endocardium, but most frequently on the cusps of the valves at or near their free border.

5. Repair. Granulation tissue replaces the fibrin, and cicatricial contraction takes place, resulting very frequently in permanent damage to the valve.

In the malignant variety of endocarditis emboli may become detached from the vegetation at any time, and float off in the blood current. These emboli invariably contain bacteria, and are known as septic emboli. Upon their lodgement in any portion of the body they form abscesses, which are known as metastatic abscesses.

Symptoms.

Simple Acute Endocarditis. As already mentioned, this disease is almost never a primary affection, but occurs as a complication of some pre-existing ailment. Hence, as in the case of pericarditis, the symptoms are apt to be masked by those of the primary disease.

As rheumatic fever is by far the most common cause of simple endocarditis, this disease is selected as the type upon which to base a detailed recital of the symptoms.

A

B

Fig. 3.—Endocarditic lesions. A, ulcerative endocarditis with perforation of one of the aortic cusps. B, healed chronic endocarditis of the mitral valve. (Hirschfelder, Diseases of the Heart and Aorta.)

With the onset of an acute endocarditis in the course of rheumatic fever, the temperature rises *without* the involvement of any new joints. The rise is not usually great, and has no distinctive characteristics, but the fever is frankly higher than has previously been the case.

Pericardial pain, usually constant and dull, may occur. The patient may experience a sense of oppression in the chest, or may complain of palpitation (consciousness of the heart-beat). Subjective dyspnea may be present. By this is meant a feeling on the part of the patient of shortness of breath, amounting sometimes to actual air-hunger, in the absence of any discoverable signs or symptoms sufficient to give rise to this feeling. It is due to the liberation and absorption of toxins.

A rise in the pulse-rate is frequently noticed, but save for this there is often found no noticeable change in the circulation. In some cases, fortunately rare, the onset of simple endocarditis is marked by the symptoms of an intense general infection, with profuse sweating, bad pulse, etc.

Course and Prognosis. Simple endocarditis usually lasts several weeks, though it is impossible to tell exactly when the infectious process has spent itself. The outlook is always grave.

Three conditions may result:

1. Death; not common; though if the patient is already greatly enfeebled by the pre-existing disease, he may be unable to withstand the added endocardial infection.

2. Recovery; may and does occur, but unfortunately not often.

3. Conversion of acute endocarditis into chronic endocarditis. This happens in the vast majority of instances.

The patient is able to withstand the infection, but the cicatricial contraction taking place in the process of repair shortens the valve cusp and permanently damages it (see Fig. 3B). Leakage then occurs, and the individual is the victim of chronic valvular heart disease.

Symptoms of Malignant Endocarditis. In considering the symptoms of this condition it is best to bear in mind that malignant endocarditis is simply general sepsis plus heart disease. Many cases occur in which no disorder of the heart can be discovered during life, and often the disease is diagnosed at first as typhoid fever.

The symptoms are many, varied, and misleading. In the main, they are those of general sepsis:

Irregular fever.
Sweats-chills.
Prostration.
Marked anemia.
Dry brown tongue.
Anorexia.
Abdominal distention.
Low muttering delirium.
Enlargement of the spleen.

Sometimes symptoms may occur referable to the heart itself, when of course, recognition of the condition becomes less difficult. As a result of the septic emboli, abscesses may occur in any part of the body.

Duration and Prognosis. Malignant endocarditis runs its course in a few weeks or at most two or three months. The outlook is hopeless.

Treatment.

Acute Simple Endocarditis. First in importance is *rest* in bed, in the recumbent position. The use of the bed-pan should be insisted upon. Rest must be main-

tained for many weeks or months in order to give the heart as little labor as possible. Patients generally find the confinement in bed long after all symptoms have disappeared as very irksome, and one of the reasons why so many cases of acute endocarditis go over into the chronic form is that patients refuse to submit to rest for a sufficient length of time.

The diet should be light, but nutritious. The patient's taste can be largely catered to. Attention must, of course, be given to the bowels and kidneys, which should be kept active.

An ice-bag over the heart is of value in quieting its action, and thus tending to relieve it of some of its work.

Drugs are not of much value. In acute endocarditis cardiac stimulants are not looked upon generally with favor, as the heart is irritated, not depressed, and does not require urging to do its work. Sometimes, if the heart shows sign of failing, stimulation is of course in order. Pain must be met by sedatives, such as codeine and often morphia.

Malignant Endocarditis. Little can be done for this distressing condition. The treatment is mainly symptomatic, and the general management is in no way characteristic. If the organism causing the infection can be discovered, treatment by means of a vaccine made from that organism (an autogenous vaccine) may prove of some aid.

ENDOCARDITIS (CHRONIC).

Etiology. Chronic endocarditis is frequently a sequel of acute endocarditis, and is, therefore, dependent upon the same causative factors as that disease (*q.v.*). It may be chronic from the start, or may be the result of a general sclerotic or fibrosing process throughout the body brought about by:

(*a*) Age.

(*b*) Arteriosclerosis.

(*c*) Syphilis.

(*d*) Alcoholism.

Nature of the Process. The valves of the heart are the structures that are damaged. Endocarditis, whether acute or chronic, practically always affects the left side of the heart; hence the valves that are the seat of the disease are either the mitral or the aortic. The cusps of the valves are the seat of a chronic inflammatory process that results in a thickening and contraction of the valve flaps. As a result one of two conditions may arise:

1. The valve cannot close completely, so that some blood leaks back through it. This is called *regurgitation*.

2. The valve cannot open sufficiently to let the requisite amount of blood pass through it. This is called *stenosis*.

There are, then, four varieties of valvular disease that commonly occur as a result of chronic endocarditis. These are in their order of frequency:

(*a*) Mitral regurgitation.

(*b*) Mitral stenosis.

(42)

(c) Aortic regurgitation.

(d) Aortic stenosis.

A few words concerning each variety will be of service:

(a) *Mitral Regurgitation.* With each ventricular systole the mitral valve should close so that all the blood in the ventricle can go into the aorta. When the mitral valve is deficient, or leaky, with each beat of the heart a portion of the blood in the ventricle is sent back through the mitral opening into the left auricle.

(b) *Mitral Stenosis.* When the mitral valve cannot open sufficiently to allow the passage of the requisite amount of blood, necessarily a portion of the blood is dammed back in the left auricle and into the four pulmonary veins, and, as the full amount of blood cannot reach the left ventricle, that chamber has an insufficient amount to deliver into the aorta at each ventricular systole.

(c) *Aortic Regurgitation.* At the completion of each ventricular systole, the ventricle is empty and the blood it contained is in the aorta. As soon as ventricular systole is over, the aortic semilunar valves should close and prevent any blood from flowing back into the left ventricle. When the aortic valves are leaky, some blood flows back into the ventricle during each ventricular diastole, thus preventing the requisite amount of blood from being delivered to the tissues with each beat of the heart.

(d) *Aortic Stenosis.* When the aortic valves cannot open sufficiently to admit the requisite amount of blood that should flow past with each ventricular systole, necessarily a portion of the blood will be dammed back in the left ventricle and an insufficient amount of blood delivered to the tissues.

The Phenomenon of Compensation.   The heart is the most adaptable organ in the body.   Were it not for its adaptability it could never withstand the sudden and unexpected strains thrown upon it at any and all times. For purposes of general discussion, the four valvular diseases under consideration can be grouped together. Each has a few special symptoms peculiar to it, and each is diagnosed by special physical signs discovered upon examination, but all four have this in common: a disorder in the mechanics of the circulation.

The changes in the valves do not take place overnight, but gradually and insidiously.   Therefore, the heart has an opportunity to accommodate itself to changing conditions, and this it does marvelously well.

When blood is leaking back through any valve, or prevented from flowing through any valve in a sufficient amount, the heart, in order to maintain the mechanics of the circulation, must obviously do two things:

1. Work faster—increased beat-rate per minute.

2. Work harder—increased power of individual contractions.

The combination of these two factors constitutes what is known as *cardiac compensation*.   The heart responds to the increased demands made upon it, and instead of the pulse-rate at rest being about 72 to the minute, the number of beats reaches 85 to 90, or even more.   Together with this, each contraction of the heart is stronger and more powerful than is normally necessary.   Just as exercise of any muscle or group of muscles causes an increase in size, so increase in the exercise of the heart causes an increase in the size of its individual muscle-fibres and in the thickness of the heart walls.   Muscle-fibres are increased in size, and new fibres make their appearance.   This change is known as *hypertrophy,* and

must be sharply differentiated from *dilatation* (presently to be mentioned) a condition in which the size of the heart is also increased.

Thus, by the combination of the two factors mentioned, the heart makes up for the leakage of blood through its damaged valve or valves, and, by driving blood more frequently into the aorta, maintains the circulatory balance. As long as this condition exists, compensation is established, and *the patient suffers from no symptoms*.

But the situation can well be likened to whipping a horse to make him run faster. For a while he responds with increased effort and increased speed, but he tires more quickly because of the excessive rate at which he is running, and because of the extra output of physical strength. So it is with the heart. For a time—months or years—the fast pace is maintained. The time inevitably comes, however, when the strain can be no longer endured, and the heart begins to show signs of fatigue. The muscle-fibres no longer contract as strongly. They stretch, and show signs of degeneration. The heart chambers become larger through this stretching, and through loss of "tone" of their walls. Then it is that the condition known as *dilatation* sets in, producing an increase in the size of the heart, but this time *not from strength,* as in hypertrophy, *but from weakness* and beginning exhaustion. As soon as this happens the mechanics of the circulation begin to fail (for the body is inexorable, and must have its proper quota of blood at all times), *symptoms make their appearance,* and compensation is said to be *failing*.

Finally, when this state of things has progressed still further, when it is all the heart can do to maintain sufficient circulation to support life, and when on all

sides the symptoms of circulatory failure are in evidence, compensation is said to be *lost*.

The Course of the Symptoms in Chronic Endocarditis. Practically all symptoms occurring, in chronic valvular disease are of mechanical origin, *i.e.,* arise from a disturbance in the normal blood-flow. They have a common cause, which can be summed up in three words: *chronic passive congestion*. As an illustration of their occurrence, may be selected a case of mitral regurgitation, the most common valvular disease, with decided failure of compensation. The description, with a few minor alterations, will fit any of the other three valvular lesions under discussion.

In mitral regurgitation the blood leaks back from the left ventricle through the mitral orifice into the left auricle, and a general damming back of the blood current, and a slowing in its rate of flow, is felt in the pulmonary veins and in the vessels of the lungs. This gives rise to a *chronic passive congestion* of the lungs, which causes a deficient aëration of the blood, resulting in (1) dyspnea and (2) cyanosis; as a result of this *chronic passive congestion* there is an excess of mucus in the air alveoli and bronchial tubes, causing irritation, and resulting in (3) cough and (4) expectoration. The damming back of the blood is next apparent in the right side of the heart, which, while perfectly sound, is not able to force the blood onward through the lungs because of the increased resistance encountered. As a result, there is slowing of the blood current, and a relative stagnation in the systemic veins, resulting in a *chronic passive congestion* of the mucous membrane of the stomach and causing (5) loss of appetite, (6) nausea, and (7) vomiting. There is also a *chronic passive congestion* of the

intestines, resulting in (8) tympanites, and of the colon and rectum, showing itself by (9) constipation and (10) hemorrhoids. The state of *chronic passive congestion* in the entire body causes an escape of serum from the blood-vessels, resulting in (11) edema. When this condition becomes more pronounced, as a result of *chronic passive congestion,* fluid appears in the abdominal cavity, and (12) ascites is present. The other serous membranes of the body (the pleura and pericardium) may be similarly affected, resulting in the conditions known as (13) hydrothorax and (14) hydropericardium. Eventually, when, as a result of universal *chronic passive congestion,* the serous cavities contain fluid, and edema is general, the condition known as (15) general anasarca is present. *Chronic passive congestion* in the brain gives rise to symptoms of (16) headache, (17) sleeplessness, (18) dizziness, (19) vertigo, and sometimes to (20) faintness and (21) periods of unconsciousness. *Chronic passive congestion* of the kidneys causes (22) scanty high-colored urine. Thus we see that practically all the symptoms of chronic valvular disease, with the exception of (23) palpitation and (24) rapid heart action, can be traced to the gradual damming back of the blood throughout the entire circulatory system, and to the resulting universal *chronic passive congestion.*

It not infrequently happens that more than one valvular lesion exists, and also very often, as compensation fails, a sound valve will become leaky—not because it has become diseased, but because, owing to the great dilatation of the heart, the ring to which the valve cusps or flaps are attached becomes stretched, and the valve cusps, though in proper working order, are not able to meet, and thus close the opening they are set to guard. A valve

that, sound itself, does not functionate properly because of dilatation of the ring to which the cusps are attached, is known as "relatively insufficient."

The pulse is usually regular while compensation is maintained, but as it fails, irregularity begins and becomes more and more pronounced, until, at the end, the heart often beats in an absolutely irregular manner, devoid of rhythm, auricles and ventricles beating when they wish, with no relation one to the other, and the condition of "delirium cordis" (delirium of the heart) is present.

In mitral stenosis the pulse is very *small, i.e.,* the vessel seems to make a small excursion against the finger, because of the relatively small amount of blood forced into the aorta. In aortic regurgitation, on the other hand, the full amount of blood is thrown into the aorta, but some leaks back into the ventricle; therefore, the pulse is very *large, i.e.,* the artery seems to be filled to the limit, and then practically emptied. This is known as the Corrigan or water-hammer pulse. Again, in aortic regurgitation, because of the rapid alternate filling and emptying of the vessels (due to the leakage back from the aorta into the left ventricle), if the skin on the forehead is rubbed with a towel so as to make it red, an alternate flushing and paling can be seen, the flushing corresponding with each beat of the heart. This is known as the "capillary pulse."

Edema is at first slight, usually noticed in the feet, ankles, and legs, and more marked if the limbs are allowed to hang down. With progressive failure of compensation, however, it grows steadily more extensive.

Dyspnea is complained of early in failure of compensation, and, in cases that do not rally, is a constant and

distressing symptom. At the last it dominates the scene, becoming extremely urgent, necessitating the sitting posture to make it bearable, and causing the "getting of the breath" to be the one absorbing idea of the patient.

Cough and expectoration are to some extent present throughout the course of failing compensation. As the end approaches, the patient is exhausted by the cough, and too weak to raise the sputum. The terminal phase is frequently edema of the lungs, the patient practically drowning in his own secretions.

Sleeplessness is a great torment, and is often most resistant to all manner of treatment. In addition to the cerebral congestion which causes it, must be mentioned other symptoms, such as dyspnea and cough which add to its severity.

Prognosis. The outlook in cases of chronic valvular heart disease is always grave. The condition is incurable, but many individuals by the exercise of care and moderation can live long and happy lives. Unfortunately, the majority have to work, and work hard, to live, and the heart cannot stand the inevitable strain. They can be patched up—once, twice, thrice—but sooner or later another failure of compensation ensues that proves fatal. Generally speaking, the outlook is better in the well-to-do than in the poor, in the exemplary than in the dissipated, and in mitral rather than in aortic disease. Prognosis also depends largely upon the faithfulness with which the patient carries out instructions, not alone as to medicines, but also as to mode of life and the avoidance of excesses of all kinds. The outlook is also largely affected by conditions of a technical nature residing within the heart, which only a physician of experience can estimate and appreciate.

4

Treatment. With compensation fully established no treatment is necessary, the patient being simply instructed to avoid violent exertion, overeating, abuse of alcohol and tobacco, and in general to live a life of moderation.

With compensation failing or lost, active treatment is necessary. The management of these cases can be grouped under several heads:

1. *Rest.* Perfect rest can, of course, never be given to the heart, but *relative* rest can, and is of the utmost importance. Bed is absolutely essential. Recumbent posture is best, for then the heart pumps blood "on the level," and not in part up-hill, as is the case if the patient is sitting up. If, however, lying down aggravates dyspnea (as is often the case), the patient can be propped up in bed on pillows. Rest in bed must be continued until, in the opinion of the physician, the heart is strong enough to stand a little added strain, when sitting in a chair and walking about the room may be allowed. The period of rest varies with each individual case, lasting from a few weeks to several months.

2. *Drugs.* Of these, digitalis, and its first cousins, strophanthus and convallaria, occupy first place. Digitalis is the best drug for a heart that is failing to compensate. The action of digitalis on a heart the victim of chronic valvular disease may be briefly summarized as follows: The effect of digitalis on the heart is due to—

(a) Effect on the *heart muscle,* which is stimulated.

(b) Effect on *vagus nerve,* which is stimulated.

The effect on the heart muscle is of two kinds—

(a) The continuous contraction or "tone" is stimulated and increased.

(b) The intermittent contraction or "beat" is stimulated and rendered more powerful.

By the action of digitalis on the tone of the heart muscle—

    I. Presure within the ventricle is decreased.

    II. Work of the heart is decreased.

By the action of digitalis on the "beat" of the heart muscle—

    I. More blood is forced into the general circulation under greater pressure.

    II. Blood is driven with more force into the coronary arteries.

    III. Blood is driven with more force from coronary arteries into coronary veins; hence the nutrition of the heart itself is greatly improved. This is an important factor in the action of digitalis.

By the stimulation of the vagus nerve the heart-rate is slowed, and diastole (the rest period of the heart) is prolonged.

Digitalis also stimulates arterial tone. Just as loading a muscle to a certain extent stimulates and increases its power of contraction, so the increased strength of the blood-current, driven through the arteries by the stimulated heart, causes the muscle-fibres in the arterial walls to contract more forcibly on the current of blood within the arteries, and to propel the blood onward more vigorously than formerly. Thus it is seen that in every way digitalis acts in a manner that is bound to secure to the tired and failing heart as much rest as possible, and at the same time in a manner to aid the arterial system in adequately carrying on the circulation.

Other drugs with a stimulating effect on the heart or on the vessels are frequently used, and at times drugs whose effect is to dilate the vessels are indicated, espe-

cially when blood-pressure is too high; but the greatest reliance is placed upon digitalis.

3. *Symptomatic Treatment.* Many special symptoms arise that necessitate attention. Dyspnea is often greatly helped by digitalis, but, until that drug has had time to show its full effect, morphia may be necessary. In the terminal stages of valvular disease morphia affords the patient the greatest relief.

Edema will often disappear to a surprising extent under digitalis, but when it is very marked, free purgation with salines in a manner similar to that described in the chapter on nephritis, is often resorted to. Ascites and hydrothorax are relieved by tapping.

Cough, until helped by the increasing strength of the heart, and consequent better circulation, will often require attention, codeine and heroin proving satisfactory.

Constipation is managed in the usual ways.

4. *Diet.* This should be bland and easily digestible. In cases with much edema it is usual to restrict the fluid intake, as is done in chronic parenchymatous nephritis (*q.v.*).

5. *Baths.* Various mineral baths, especially those of Bad Nauheim, in Germany, enjoy a great reputation for their beneficial action on cases of chronic valvular disease. The baths probably help because they tend to deplete the system of toxic products, and the entire "cure" aids mainly because the patient is away from home, business cares and worries, and able to devote 24 hours a day to the task of recovery. Nauheim baths, as far as their chemical composition is concerned, can be duplicated in the tub at home, but the results are not so good as those seen in the health resort.

6. *Exercise.* When the heart is again fairly well compensated, carefully graduated exercises are of great benefit in increasing the strength and endurance of the heart muscle. These exercises consist usually in various movements of arms, legs, and body against a certain amount of resistance offered by the nurse. They must be explained and illustrated in detail by the physician, and must be carried out with great care, as their abuse may lead to irreparable harm.

CHAPTER VIII.

MYOCARDITIS AND ANEURYSM OF THE AORTA.

Myocarditis.

Myocarditis, as the name indicates, is an inflammation of the myocardium or heart muscle itself. The disease may be acute or chronic. Cases of acute myocarditis hardly ever occur primarily, but almost always as a complication of an acute infectious disease such as diphtheria, typhoid fever, pneumonia, or rheumatic fever. Hence, acute myocarditis is almost invariably of toxic origin. The fibres of the heart muscle are swollen, show evidences of granular degeneration, and their striations are blurred and indistinct.

Acute Myocarditis.

Symptoms. There may be very few, or almost none. Frequently, however, there is

1. Pallor of the face, which is striking and persistent.

2. Vomiting.

3. Weakness and listlessness not accounted for by the primary disease from which the patient may be convalescing, the patient giving the impression of being profoundly ill.

4. Rapid pulse, not particularly marked, the rate being usually about 100. There may or may not be irregularity of the pulse.

5. Feeble and unstable pulse, a very slight exertion being sufficient to send up the pulse-rate out of all proportion to the degree of effort.

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The course of the disease is variable. Death may occur within two or three days. The presence of the condition may be unrecognized, and sudden death occur, or alarming symptoms may alternate with periods of almost perfect well-being. The outlook is always grave if the degree of myocarditis is at all advanced.

Treatment. There is no measure that will directly influence the damaged heart muscle favorably. Proper treatment for the pre-existing disease is, of course, an essential.

As soon as signs and symptoms of myocarditis are detected, "the indication is to maintain the patient in absolute repose of mind and body. Physical effort is dangerous, and so long as cardiac weakness exists the patient must remain in bed. He should receive as much highly nutritious and simple food as he can assimilate—milk, eggs, broth, etc. The bowels are to be kept active, though depleting purgatives are to be avoided. Strychnine is highly serviceable. . . . In conclusion, it may be repeated that the agencies of greatest service are : rest, food, strychnine, and stimulants, in the order named" (Babcock).

CHRONIC MYOCARDITIS.

Etiology.

1. Degenerative changes in the coats of the arteries (arteriosclerosis) — by augmenting resistance to the heart.
2. Chronic nephritis, both parenchymatous and interstitial —
3. Hard toil.
4. Poor quality of blood.

(*a*) Cancer.

(*b*) Chronic suppurations.

(*c*) Anemia.

(*d*) Chronic diarrhea.

(*e*) Insufficient food.

5. Toxins of acute infectious diseases.

Symptoms. These are notoriously uncertain. Often there are no symptoms, the heart doing its work fairly well until of a sudden it stops, exhausted, and the patient falls dead. Heart action may be feeble and irregular; there may be dyspnea, edema, and all the symptoms detailed under failure of compensation in the section on chronic endocarditis, resulting from a combination of failing heart muscle and dilatation of all the chambers of the heart.

Osler has grouped cases of chronic myocarditis in the following practical manner:

1. Those in which sudden death occurs with or without previous indications of heart trouble.

2. Cases in which there are cardiac arrhythmia, shortness of breath on exertion, attacks of asthma, collapse symptoms, with sweats and extremely slow pulse.

3. Cases in which there are cardiac insufficiency and symptoms of dilatation of the heart.

The outlook in cases of chronic myocarditis is very grave. The heart is permanently damaged, and in addition there is the ever-present action of the exciting cause, whatever it may be, so that, while recovery from attacks of heart failure frequently occurs, their recurrence is to be expected, and eventually a fatal seizure is bound to come.

Treatment. Rest is first in importance for the patient—prolonged rest both of mind and body. Every ex-

ertion must be prevented, as the heart needs freedom from all possible strain in order to recuperate. Restlessness is well acted upon by morphia. Strychnine has found much favor in toning up the heart. Stimulants, especially aromatic spirits of ammonia, are often very useful. The diet must be light, and the amount of fluids must frequently be restricted, especially in cases with edema. As in the case of acute myocarditis, the bowels must be kept well open, but the patient's strength must not be sapped by the use of drastic purgatives. No set rule can be given for the management of these cases, as the frequently complicating nephritis and arteriosclerosis make of each case an individual problem. The nurse must be ever watchful for signs of returning failure on the part of the heart, for lack of proper elimination on the part of the kidneys, and in the control of the patient's daily life must err on the side of caution, for any excessive exertion may at once destroy all the advantage gained by weeks of patient and unremitting care.

ANEURYSM OF THE THORACIC AORTA.

By aneurysm is meant dilatation of an artery. Aneurysms may occur in any artery of the body, but this section concerns itself solely with aneurysm of the aorta. Aortic aneurysms vary in size from that of a walnut to that of a child's head.

Etiology. Aneurysms originate from the gradual giving way of the aorta, owing to disease of the wall of the artery, especially of the media or middle coat. Factors entering prominently into the formation of aortic aneurysms are:

1. Arteriosclerosis. Almost always present.   .

2. Syphilis. Now believed to be a factor in the causation of the vast majority of aortic aneurysms.

3. Age. Usually occurs in individuals over 40.

4. Sex. Males affected eight times as frequently as females.

5. Alcoholism and occupations involving great physical exertion.

Symptoms. These are very varied, as can readily be imagined when it is borne in mind that the sac, or tumor, growing from the aorta, may spread in any direction. Most of the symptoms produced by aneurysm are due to pressure upon adjacent structures, such as the lungs, trachea, esophagus, ribs, and various nerves running through the chest. Thus it will be seen that to describe all symptoms would necessitate an anatomical discussion as to the relationship of the thoracic contents, and mention of every direction in which an aneurysm could exert pressure. This would involve an amount of detail that is obviously beyond the scope of a short lecture.

There are, however, certain general features shared to a greater or less extent by all aneurysms, whatever their position along the course of the thoracic aorta, and these will now be briefly considered:

1. Pain. One of the earliest and most constant symptoms. Its intensity depends upon the direction in which the sac presses. It is described as boring, grinding, cutting, burning, etc. It is apt to be very constant, unlike chest pains arising from causes other than the growth of a tumor. It may be aggravated or lessened by a change in position, according as pressure upon the intercostal nerves is increased or lessened.

2. Dyspnea. Very common, but varies much in severity. Most marked when the aneurysmal sac presses upon the trachea, large bronchi, or lungs.

3. Cough. Common, but also very variable. May be slight, or at times the most distressing symptom. When due to pressure on the trachea, the cough has a harsh quality, known as "brassy." Sometimes cough is hoarse, due to paralysis of a vocal cord.

4. Expectoration. Usually associated with cough.

5. Hemoptysis. This frequently occurs, the blood coming from raw areas in the bronchi, from lung destruction due to pressure, or from the sac itself, in which case it is spoken of as "weeping aneurysm."

6. Dysphagia. Caused when the tumor presses upon the esophagus. Swallowing may be moderately or extremely painful, and it may gradually become impossible for food to pass into the stomach.

7. Perceptible tumor. At times the sac projects outward, eats its way through the ribs, and bulges from the front of the chest.

Course of the Disease and Prognosis. The course is usually lengthy, the tumor gradually growing larger, and causing more and more pain and suffering through the constantly increasing pressure. The outlook is bad. Occasionally a small sac will stop growing, and a condition of arrest be brought about, but the walls of the sac are the walls of a diseased vessel, and under the constant strain of pressure from the contained blood they usually end by giving way. Death occurs sometimes by rupture of the sac, with immediate profuse hemorrhage, or else as a result of the mechanical interference with respiration or circulation, from exhaustion, or from starvation, as when the esophagus is obliterated by pressure.

Treatment. This is very unsatisfactory. Attention must be directed to the underlying cause, when discoverable, and in syphilitic patients salvarsan and mercury are used extensively.

Rest in the recumbent position is essential, if this position does not aggravate any of the symptoms. Attempts must be made to reduce arterial tension, and to lessen the volume of blood. Tufnell, of Dublin, has recommended a very rigid and restricted diet, with which good results have been obtained. His dietary consists of 2 ounces of bread and butter with 2 ounces of milk for both breakfast and supper, and 2 to 3 ounces of meat and 3 to 4 ounces of milk for dinner. Few patients have the hardiness to persist with such a diet any considerable time ; but food restriction, and particularly fluid restriction, are important.

Among drugs, iodide of potassium holds first place. It is usually administered in moderate doses, and frequently has a most beneficial effect upon pain.

The remainder of the treatment is symptomatic, and in the vast majority of cases morphia will have to be freely resorted to before death brings relief from suffering. Various surgical procedures have been attempted, all of doubtful value.

CHAPTER IX.

BLOOD-PRESSURE.

By "blood-pressure" is meant the amount of pressure that the blood is under in the arteries. While in physiological experiments venous and capillary pressure are also determined, in the actual practice of medicine the arterial pressure is the only one whose determination is in general use. The importance of blood-pressure lies not in the pressure the fluid blood itself is under, but in the information it gives with regard to the amount of pressure the arterial walls have to resist, and the amount of resistance the heart has to overcome. A nurse will not be required to estimate blood-pressure, but she will see it done so often, and hear the results of this method of examination discussed so frequently, that she should know enough of the subject to appreciate its value and significance. Moreover, there are several diseases so dependent upon and so intimately associated with variations in blood-pressure that their proper understanding is impossible without an appreciation of the significance of arterial tension.

The instrument employed for the determination of blood-pressure is known as the sphygmomanometer. There are many varieties on the market. All possess a cuff, made usually of cloth or leather, lined with a rubber bag that can be inflated with air through a tube by means of a little pump. This bag is also connected by another tube with a column of mercury running on a scale graduated in millimeters, the unit of estimation of blood-pressure being a millimeter of mercury. Thus, if

a certain pressure is said to be 160, 160 millimeters of mercury is meant. When the so-called "systolic" blood-pressure is to be determined, the cuff is attached to the upper arm, with the rubber bag lying next to the skin and covering the inner side of the arm where runs the brachial artery. The bag is then filled with air by means of the pump until enough constriction is exerted to obliterate the pulse at the wrist. During this procedure the column of mercury rises rapidly, due to the pressure exerted upon it from the air within the bag. When the radial pulse can no longer be felt, a small thumb-valve in the pump is slightly opened, letting any desired amount of air escape from the bag. Air is gradually allowed to escape until the radial pulse can again be felt because of the lessening of compression over the brachial artery. The reading on the scale of the mercury column at the time the *first faint beat* can be felt at the wrist constitutes the *systolic pressure*.

Latterly there has come into use the "auscultatory" method of blood-pressure estimation, by which means both systolic and diastolic pressure (to be mentioned presently) are determined. This method will not be described, as it is rather complicated, and would be of no practical use to the nurse.

There are five factors which go to maintain normal blood-pressure:

1. The energy of the heart.

2. The resistance offered to the heart by the passage of the blood through the arteries.

3. Vasomotor tone.

4. Volume of blood.

5. Viscosity of blood. When blood is watery, pressure is invariably low.

Most of these factors are self-evident, but No. 3 (vasomotor tone) requires a word of explanation. By vasomotor tone is meant the average general tonus or steadily maintained pressure of the arterial walls upon the blood running within them. This tonus may, and does, vary greatly in normal persons at different times and in different parts of the body, depending upon the particular needs of the particular organ or tissue at any particular time. The mechanism of vasomotor tone is reflex in origin, and is governed by the sympathetic nervous system. Sympathetic nerve-fibres run in the arterial walls, some being called vasoconstrictors, *i.e.*, causing the muscle fibres in the walls of arteries to contract, and thus to *narrow* the calibre of the vessel; others known as vaso-dilators, causing the muscle-fibres in the walls of the arteries to dilate, and thus to *widen* the calibre of the vessel. Thus, during digestion, due to vasodilator action, the blood-vessels of the intestinal walls dilate, and more blood is brought to the parts. Again, during the active exercise of any muscle or group of muscles the vessels within the muscles dilate in order that more blood may be supplied. Application of cold, on the other hand, causes vasoconstriction and a blanching or pallor of the skin. Vasomotor tonus and vasomotor action are the great equalizers of blood-pressure all over the body, and one of the most important factors in maintaining an even circulation throughout the body, the various portions of which are subjected to such diverse and unexpected demands.

Blood-pressure is divided into two chief phases:

1. *Systolic Pressure.* This term is applied to the blood-pressure within a given artery, when the greatest force is exerted within it, *i.e.,* during ventricular systole.

2. *Diastolic Pressure.* This is the degree of pressure exerted within an artery during cardiac diastole, and represents the lowest pressure occurring in the vessel.

The so-called *pulse-pressure* is obtained by subtracting the diastolic pressure from the systolic pressure, and denotes the total variation in pressure occurring during a cardiac cycle, thus:

Systolic pressure .... 145 mm. of mercury.
Diastolic pressure 100   "   "   "

Pulse pressure 45   "   "   "

Blood-pressure is influenced normally by a variety of factors, some of which are here briefly mentioned:

(*a*) Age. Low in childhood, gradually rising with advancing years.

(*b*) Sex. Slightly lower in women than in men.

(*c*) Digestion. Higher during its greatest activity.

(*d*) Muscular development. Higher in those well developed.

(*e*) Mental worry or fatigue. Lowered.

(*f*) Altitude. Slightly lowered.

Normal Blood-pressure. Janeway, in more than 2000 blood-pressure determinations, has found the high normal limit of systolic pressure, with very few exceptions, to be 145 mm.; his figures for women are 10 mm. less. The same authority believes normal diastolic pressure to be from 25 to 40 mm. below the systolic pressure.

As a general guide for the estimation of normal systolic pressure, Faught has formulated the following rule: "Consider the normal average systolic pressure at the age of 20 to be 120 mm. of mercury; then for each year of life above this add $\frac{1}{2}$ mm. to 120." Thus, for a man of 50 the rule would read: $120 + 15$ ($\frac{1}{2}$ of 30) $= 135$ mm. of mercury as normal systolic pressure.

Abnormal Blood-pressure. Abnormal blood-pressure can be classified under two heads:

1. Hypotension—lowered blood-pressure.

2. Hypertension—heightened blood-pressure.

1. *Hypotension* occurs in connection with the following conditions:

(*a*) Approaching death.

(*b*) Mitral stenosis.

(*c*) Paroxysmal tachycardia.

(*d*) Shock and collapse.

(*e*) Hemorrhage, external or internal.

(*f*) Infections, especially—

    I. Tuberculosis.

    II. Typhoid or any continued fever.

    III. Cholera or any severe diarrhea.

(*g*) Any wasting condition—

    I. Cancer.

    II. Pernicious or severe secondary anæmia.

(*h*) Diabetes.

(*i*) Neurasthenia.

Effects and Danger of Hypotension. "The direct effect of a falling blood-pressure is the accumulation of an abnormal amount of blood in the veins, and a slowing of the current in the arteries. This will affect the capillary circulation, and interfere with the nutritive and secretory processes which depend upon it. The most serious effect is on the heart, as it has been shown that complete loss of vasomotor tone soon leads to death, because of the gradual accumulation of nearly all the blood in the body on the venous side, so that the heart has no blood upon which to act" (Faught).

2. *Hypertension.* This condition is an accompaniment of two of the most frequent chronic diseases of middle

and old age—arteriosclerosis and chronic interstitial nephritis.

A condition of hypertension exists when the systolic pressure is over 160 mm. of mercury. When the systolic reading reaches 200 mm. the condition is serious, and when over 200 mm. it becomes dangerous, though many individuals are met with that enjoy relatively good health together with an alarmingly high blood-pressure. There are many combinations of and relationships between the systolic and diastolic pressures, which will not be discussed here, as they belong to the province of the physician rather than to that of the nurse.

Hypertension is usually present in connection with the following conditions:

    (*a*) Arteriosclerosis.

    (*b*) Chronic interstitial nephritis.

    (*c*) Cardiovascular-renal disease.

    (*d*) Apoplexy.

    (*e*) Acute nephritis.

    (*f*) Chronic parenchymatous nephritis.

    (*g*) Uræmia.

The management of both abnormally low and abnormally high blood-pressure will not be entered into here, as it is dealt with in connection with the diseases in which those conditions occur.

CHAPTER X.

THE URINE.

THE urine is both an excretion and a secretion. It is an excretion in the sense that its component parts are no longer of any use to the body and hence must be eliminated, and it is a secretion in the sense that it is the product of the activity of a gland, the kidney.

Normal Urine. Always aqueous. Usually transparent, though it may be clouded by—

Mucus, earthy phosphates of calcium and magnesium, or by urates, these last usually giving a "brick-dust" sediment.

Color. Pale lemon-yellow to reddish-brown.

Reaction. Usually acid, due to the presence of acid phosphates of sodium and calcium. The acidity of the urine varies at different times. In the early morning it is highest. After meals, *i.e.,* during the period of greatest digestive activity, urinary acidity is lowest. If food is mainly vegetable and rich in alkaline salts, the urine may become neutral or even alkaline.

Specific Gravity. Varies from 1.012 to 1.024. The early morning urine shows the highest specific gravity.

Amount in Twenty-four Hours. Roughly speaking, 1500 c.c.—3 pints or 50 ounces.

The amount of urine is affected by several factors—

1. Intake of fluids. The amount of urine is increased proportionately to the amount of liquid drunk, and dis-

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proportionately when some of the fluids taken have a markedly diuretic action, *e.g.,* beer, coffee.

2. Intake of food. If a large amount of solid food is taken, together with a relatively small amount of liquid, the urine will be decreased in amount.

3. Digestion. The amount of urine is at its greatest a few hours after a meal, and at its lowest during the early morning hours.

4. External temperature:
    1. Cold. Amount of urine greatly increased, and specific gravity lowered, because the skin, which is the other great channel for the elimination of fluids, is not active. We do not perspire freely in winter.
    2. Heat. Amount of urine decreased, and specific gravity raised, because of increased fluid elimination through the skin.

5. Exercise. Increases amount of urine because of increased metabolism going on throughout the body.

6. Drugs. Some increase urinary flow; others decrease it.

Composition of Normal Urine. In a twenty-four-hour specimen totalling 1500 c.c. there will occur about 72 grams of solids.

These solids and their approximate proportions in grams are as follows:

	Gms.
Urea	33.18
Uric acid (urates)	0.55
Hippuric acid	0.40
Creatinin, xanthin	
Hypoxanthin, guanin	11.21
Ammonium salts	

Inorganic salts:

    Sulphates, phosphates and chlorides of sodium⎫
        and potassium ....⎪ Gms.
    Phosphates of calcium and magnesium .... ⎬ 27.00
Organic salts:⎪
    Acetates ....⎭
    Sugar. Trace.

<center>Gases. N and CO_2.</center>

(This table is given merely to show what an exceedingly complex substance urine is).

A few words should be said about *urea*. This is the most abundant organic constituent of urine. Interest in this substance centers in the fact that it is the chief end-product of proteid metabolism. Proteid is the substance that is most difficult of elimination for the kidneys. Therefore estimation of the amount of urea eliminated can indicate to quite an accurate degree the state of kidney efficiency. Of course, in order to properly estimate the urea output, the exact amount of proteid intake must be accurately known, otherwise the urea estimation is obviously quite useless.

The estimation of *uric acid* and of *creatinin* are frequently made nowadays, not because of the intrinsic importance of these substance, but because, like urea, they indicate the amount of proteid elimination on the part of the kidney.

Abnormal Urine:

    Color. Pale in diabetes.

           Pale in hysteria.

           Pale in chronic interstitial nephritis.

           Deep brown or almost red in practically all acute fevers and in acute nephritis.

           Dark in liver disease and jaundice, due to bile pigment.

           Brown to bright red when containing blood.

Changed by drugs:

Blue after taking methylene blue.

Brown-smoky after taking carbolic acid.

Bright yellow after taking santonin.

Amount in Twenty-four Hours. Usually considered pathological when under 500 c.c. (1 pint) or over 3000 c.c. (3 quarts).

Small output of urine is known as *oliguria.* Oliguria occurs in—

1. Cardiac disease, with low blood-pressure.

2. Acute fevers.

3. Acute nephritis and chronic parenchymatous nephritis.

4. Cholera and all severe diarrheas.

5. Eclampsia and uræmia.

(Obstructions of a mechanical nature, tumors, etc., are not here considered).

Large output of urine is known as *polyuria.* Polyuria occurs in—

1. *Diabetes mellitus.*

2. Diabetes insipidus.

3. During absorption of large effusions:

(*a*) Pleural.

(*b*) Peritoneal.

4. Convalescence from typhoid fever and from other acute infections.

5. *Chronic interstitial nephritis.*

6. Exophthalmic goitre (Graves' disease).

Albuminuria, *i.e.,* albumin in the urine. The presence of albumin in the urine is almost always a pathological finding, though its mere presence by no means signifies kidney disease.

Albumin in the urine may be due to the presence of

pus from a cystitis, or to the presence of blood from a hemorrhage somewhere in the urinary tract.

There are so many causes of albuminuria, apart from disease of the kidneys, that it will be worth while to mention some other conditions under which this condition can and does occur.

Albuminuria may be—

1. Cyclic. Appearing, disappearing, and reappearing at certain definite intervals. Usually of no known significance.

2. Dietetic. Appearing after a meal over-rich in proteid.

3. Febrile. Due to degenerative changes in the kidney taking place during the height of the acute fevers. These changes are usually transitory, and complete recovery is the rule.

4. Toxic. Poisoning by any substance, especially ether.

Glycosuria, *i.e.,* sugar in the urine. Sugar in the urine is a far rarer condition than is the presence of albumin. Sugar may appear in a transitory manner after meals rich in sugar, but persistent glycosuria is always pathological. In the large majority of cases the cause of persistent glycosuria is diabetes mellitus.

Indicanuria, *i.e.,* excessive amount of indican in the urine. This condition occurs when an undue amount of toxic material is being absorbed into the body from the intestinal tract.

Pyuria, *i.e.,* pus in the urine, may occur as the result of a urethritis, a cystitis, or as a result of a kidney abscess, a pyelonephritis, or tuberculosis of the kidney.

Some Suggestions for the Collection of Urinary Specimens. When taking charge of a case it is well

for the nurse to have a specimen of urine ready for the physician at his visit on the following day, even if a request for it has not been made. Urinary specimens may be divided into three classes:

1. Specimen of mixed urine.
2. Specimen of twenty-four-hour urine.
3. Catheterized specimen for special examinations.

Classes 2 and 3 will be specially requested by the physician. Where "a specimen of urine" is asked for, the nurse can take it for granted that "mixed" urine is meant—*i.e.,* some passed in the evening mixed with some passed in the morning. Save when specially requested, or when the output of urine is exceedingly scanty, a specimen should not consist entirely of urine passed at one voiding. If a twenty-four-hour specimen is requested, the nurse should always ask the physician if he wishes the entire quantity, or whether a portion of the total urine will suffice; and the number of ounces passed in the twenty-four hours should always be plainly recorded on the label accompanying the specimen.

A four-ounce bottle should be used for the specimen of urine, and, unless impossible to do so, the bottle should be entirely filled. There are few things more irritating to the physician than to have specimens of urine totalling from half an ounce to an ounce and a half received from patients passing fifty times that amount in twenty-four hours; and yet these "homeopathic" specimens are constantly being sent to the laboratory. If there is plenty of urine, let the specimen be generous. The bottle should, of course, be clean—not necessarily sterile, unless the physician particularly requests this. It should be firmly stoppered with a well-fitting cork. Fruit jars, sal hepatica jars, Pluto water bottles with their little tin

caps for corks, are all unsuitable for urinary speci-
mens. The nurse must be certain that the previous con-
tents of the bottle have all been done away with, as in
the author's experience one case is recalled where a
marked sugar reaction was obtained, which was subse-
quently traced to the specimen bottle having contained
some substance rich in glucose.

The name of the patient, the date, the total amount of
urine if the specimen is a "twenty-four-hour specimen,"
and the hours of voiding, if the specimen is "mixed,"
should all be plainly written on a label, which should be
pasted on the bottle. It may seem superfluous to men-
tion all these details, but their enumeration is the result
of several years' experience in the laboratory with all
manner of specimens, containers, legible labels, illegible
labels, and, last of all, no labels!

The nurse should be sure to deliver or send all speci-
mens *promptly* to the physician's laboratory. If she
thinks there may be some delay, she should ask the
physician what to add to the urine as a preservative. A
small piece of thymol or a few cubic centimeters of
chloroform are commonly used. Urine rapidly decom-
poses, and "stale" urine is unfit for examination.

Urinary Examination. It is not within the province
of the nurse to examine urinary specimens. There are,
however, four tests in connection with urinary analysis
that any nurse should be able to perform satisfactorily,
and these tests are so common that she should be
familiar with them:

    1. Determination of the reaction of the urine.

    2. Determination of the specific gravity of the urine.

    3. Determination of the presence or absence of
       albumin.

4. Determination of the presence or absence of sugar.

1. *Determination of the Reaction of the Urine.* Dip a piece of *blue* litmus paper in the urine. If it turns *red,* the urine is *acid.* If it does not change color, dip a piece of *red* litmus paper in the urine. If it turns *blue* the urine is *alkaline.* If it does not change color, the urine is *neutral.*

2. *Determination of the Specific Gravity of the Urine.* Pour urine into a cylindrical jar made for the purpose, or into a 100-c.c. graduate. Fill the jar or graduate to within an inch of the top. See that no bubbles have formed on the surface of the urine. Drop the urinometer or specific gravity float into the urine with a spinning motion. Allow it to settle and read the specific gravity on the scale of the urinometer, reading at the *bottom* of the meniscus. The meniscus is that portion of the fluid that appears to be "climbing up" the sides of the graduate. This takes place because of capillary attraction. The reading of the urinometer scale should be made at the level of the fluid, which is often appreciably below the top of the meniscus.

3. *Determination of the Presence or Absence of Albumin.* (*a*) Fill a small test-tube two-thirds full of urine. Hold it at its lower end, and boil the upper inch of urine in the Bunsen burner or alcohol flame. If *no cloud appears* in the urine, albumin is *absent.* If a cloud appears, add two or three drops of 3 per cent. acetic acid. If the cloud disappears it is due to phosphates. If it persists and grows more dense on boiling again, albumin is *present.*

All variations may exist in the amount of cloud obtained, depending upon the amount of albumin. The

faintest film may be seen, or the urine may boil almost solid.

(b) Pour about 2 c.c. of cold nitric acid (HNO_3) into a small test-tube. With the aid of a dropper allow about 1 c.c. of urine to flow *slowly* down the side of the test-tube and to overlay the nitric acid. In the presence of albumin a *white ring* is seen at the point of contact of nitric acid and urine.

4. *Determination of the Presence or Absence of Sugar*. Fehling's solution is used for this test. It consists of two elements:

I. A solution of copper sulphate.

II. A solution of rochelle salts.

To perform the test: In a small test-tube place 1 c.c. of solution I. Add 1 c.c. of water. Then add 1 c.c. of solution II, and 1 c.c. of water. Bring to a boil. Add 1 c.c. of urine and boil again. If the solution remains a clear beautiful blue, sugar is absent. If it turns to a dirty green, or to a reddish-yellow, or to an actual red, sugar is *present*.

Further urinary tests will not be mentioned here, as it is felt that they lie outside the sphere of the trained nurse, unless she wishes to become a laboratory worker.

CHAPTER XI.

URÆMIA.

URÆMIA is not a disease in itself, but a condition that occurs both alone and as a complication of many diseases. Thus, we often say that such-and-such a patient is doing badly—"he is becoming uræmic"—meaning thereby that the condition recognized as uræmia is setting in.

The cause of uræmia remains as yet unknown. There are many theories advanced, but none has hitherto met all requirements. It is definitely known that uræmia is of toxic origin, and arises from failure on the part of the body to properly eliminate its waste-products. It has been claimed by some that uræmic patients are those whose urine has lost its toxicity. Urine should be toxic because of the waste-products it contains; when it loses its toxicity, the waste-products are not excreted, and hence are stored up in the body.

Acute Uræmia. A typical attack of acute uræmia may appear without any previous signs of illness, or else the condition may appear as the terminal factor in many diseases, especially chronic interstitial nephritis.

There may be some headache and dizziness, though these are often slight. The most striking feature of an attack of acute uræmia is the *convulsion*. It is both tonic and clonic in character, the patient being alternately rigid and throwing himself about, and often frothing at the mouth. The pupils are usually dilated and active. There is usually a variable period of unconsciousness fol-

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lowing the convulsion, which resembles in many ways an epileptic fit.

During the attack of acute uræmia there is usually complete or partial suppression of urine; and during the entire duration of uræmia, whether acute or chronic, the amount of waste-products excreted by the kidneys is below par, even though the total amount of urine may be well up to normal.

After the convulsive seizure blindness may occur, which may persist for several days. At times coma may develop without any convulsion occurring. Nausea and vomiting may be prominent symptoms.

Chronic Uræmia. By chronic uræmia is meant that group of symptoms which denote insufficient elimination. These symptoms may persist for a long or short time, may clear up entirely under treatment, or at any moment the patient may be thrown into an attack of acute uræmia. Chronic uræmia hardly ever appears at the first sign of ill health; it is almost always a complication of some pre-existing ailment, most frequently of chronic nephritis. It is important for the nurse to be familiar with some of the manifestations of chronic uræmia, as thus she can observe changes in the patient, and, what is more, can appreciate their significance.

The symptoms to which chronic uræmia gives rise will be mentioned under the headings of the various systems of the body:

1. *Cutaneous*. Itching. May be only slight or else universal and intense. There may be many varieties of skin eruptions.

2. *Respiratory*. Dyspnea, which may be

  (*a*) Continuous.
  (*b*) Paroxysmal.

(*c*) Cheyne-Stokes. A period of moderate breath-
ing followed by a period of gradually increas-
ing deep breathing, which in turn gradually
fades away, and is followed by a period of
very shallow breathing.

3. *Circulatory.*
   (*a*) High arterial tension. Very commonly occurs,
   but its absence is by no means invariably a
   favorable sign.
   (*b*) Heart failure. Usually myocardial degenera-
   tion (see chapter on Myocarditis). Common.
   Many deaths from this cause.
   (*c*) Rather slow pulse. May be some irregularity.
   (During convulsive seizures of acute uræmia
   pulse may be small, soft and rapid.)

4. *Nervous.*
   (*a*) Convulsions. These have been described. In
   chronic uræmia there may be merely muscular
   twitchings which never reach the dignity of
   a convulsion.
   (*b*) Dimness of vision. A sense of a film before
   the eyes. An important symptom of advanc-
   ing chronic uræmia.
   (*c*) Gradually increasing mental dullness, eventually
   passing into semi-consciousness and coma.
   (*d*) Headache and giddiness. Very frequent and
   very important. Their increase is a bad sign,
   and their lessening a good sign.

5. *Gastro-intestinal.*
   (*a*) Loss of appetite.
   (*b*) Nausea and vomiting. May be the first symp-
   tom that attracts attention. May be slight
   or very intense.

(*c*) Hiccough. Violent and persistent. A very bad sign.

(*d*) Constipation.

(*e*) Diarrhea. Usually occurs only in last stages.

Prognosis. The outlook in uræmia is always grave. Much depends upon the condition existing before uræmia developed. Acute uræmia developing in the presence of an acute nephritis offers a better chance for recovery than acute uræmia developing on top of a chronic parenchymatous or chronic interstitial nephritis. Chronic uræmia is always serious, and, as in the case of acute uræmia, the outlook depends largely upon the pre-existing condition. Usually, complete recovery from chronic uræmia is not possible, but often considerable improvement can be obtained and maintained for long periods of time.

Treatment. "The correct treatment of nephritis is the best way of escaping acute or chronic uræmia."

With the exception of the management of the convulsion, for which chloroform is often necessary, the treatment of acute uræmia is identical with that of acute nephritis, and therefore will be dealt with in connection with that disease.

The treatment of chronic uræmia is identical with that of chronic parenchymatous or chronic interstitial nephritis, and will be dealt with under those headings.

CHAPTER XII.

NEPHRITIS (BRIGHT'S DISEASE).

NEPHRITIS signifies inflammation of the kidney. It does not include the surgical conditions of that organ, such as tuberculosis, kidney stone, kidney abscess, etc.

There are many classifications of nephritis, some of them very complicated. The following is simple, and, from the practical standpoint, satisfactory:

1. Acute nephritis.
2. Chronic nephritis.
   (*a*) Chronic parenchymatous nephritis.
   (*b*) Chronic interstitial nephritis.

ACUTE NEPHRITIS.
(ACUTE BRIGHT'S DISEASE.)

Acute nephritis is a diffuse inflammation of the kidneys, involving practically every portion of the kidney structure, and brought on by a variety of agencies.

Etiology.

1. Infectious diseases, especially:
   (*a*) Scarlet fever. By far the most important. The toxin of scarlet fever seems to be particularly injurious to the kidneys.
   (*b*) Diphtheria.
   (*c*) Tonsillitis.

Acute nephritis may occur as a complication of *any* infectious disease.

2. Toxic agents:
   (*a*) Drugs, *especially* cantharides, turpentine, phenol, salicylic acid, mineral acids, alcohol, chloroform, mercury.

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(*b*) Extensive burns. Acute nephritis occurs in these cases as a result of toxic material formed from the destruction of the skin.

(*c*) X-ray.

(*d*) Acute gastro-intestinal disorders.

(*e*) Disorders of metabolism.
Diabetes.
Gout.

3. Cold, especially when combined with getting wet.

4. Pregnancy, because of added strain on kidneys, through having to excrete for two individuals.

Pathology. The kidneys are usually enlarged, and may be slightly edematous. The capsule peels off with ease. The surface is pale, and minute hemorrhages may be visible.

Microscopically the *convoluted tubules* are most affected. The lining cells are swollen, cloudy, or granular, and their nuclei stain badly or not at all.

In the lumen of the tubules may be seen hyaline or granular casts, droplets of fat, red blood-cells and some leukocytes. The *glomeruli* are also affected. The cells lining Bowman's capsule are degenerated and there may be hemorrhages into the capsule.

Symptoms. Acute nephritis usually comes on suddenly. At times there may be an initial chill, with some fever (rarely over 102°), headache, drowsiness, nausea and vomiting. The characteristic features of acute nephritis, however, can be grouped under three headings:

(1) Edema.

(2) Urinary changes.

(3) Uræmic manifestations.

1. *Edema.* The edema of acute nephritis is different from any other edema. It usually comes on rapidly, the

patient having a rather pasty and puffy appearance. The edema is often almost universal, not very marked at first, decidedly firm and pitting but little on pressure. After having persisted for a time it is, of course, more notice-able in the dependent portions of the body. It may be slight or may assume enormous proportions.

2. *Urinary Changes.* The urine is always scanty (4 to 10 ounces in twenty-four hours), or there may be com-plete suppression of urine. This latter condition can exist for two or three days and recovery still be possible; but persistent anuria is always a most serious sign. When urine is obtainable it is turbid, smoky, and of a reddish-brown color. Its reaction is acid, the specific gravity very high, from 1.025 to 1.035 (though in exceptional cases it may be as low as 1.018), and albumin is present in large amounts. Microscopically there is seen much epithelium and many red blood-cells. Casts of all varie-ties—blood, epithelial, granular, and hyaline—are present in great abundance. One of the best signs of improve-ment in a patient with acute nephritis is an increase in the output of urine, and until this occurs no case can be looked upon as doing satisfactorily.

3. *Uræmic Manifestations.* Some of these, such as headache, drowsiness, nausea, and vomiting, have al-ready been referred to in connection with the onset of acute nephritis. At any time *convulsions* may occur, to be followed, in the most severe cases, by coma. The convulsions are similar in every respect to uræmic con-vulsions. The pulse may be fast or slow, and is usually of high tension.

Course of Disease. As a rule, if the patient is not overcome by the initial shock of the attack, improvement begins in two or three days, first shown, as stated above,

by an increase in the output of urine. With this increase there is also a corresponding decrease in the amount of edema, and a lessening of the uræmic manifestations. Acute nephritis is usually a brief disease. If it maintains its greatest intensity, death must come within a few days; if it lessens in severity, the picture becomes one of chronic parenchymatous nephritis, to be presently discussed.

Prognosis. This is always grave. There are three possibilities confronting the patient:

1. *Death,* which occurs in a fair proportion of cases and is due, usually, to—

(*a*) Uræmia (convulsions and coma).

(*b*) Persistent anuria.

(*c*) Inflammations of respiratory tract.

2. *Recovery,* which occurs in a large percentage of cases, especially when the intensity of the acute stage is of short duration, so that no portion of the kidney structure is permanently damaged.

3. *Chronic nephritis,* which is the fate of many patients that are able to rally from the acute stage, but whose kidneys have suffered permanent and incurable structural changes.

The outlook in acute nephritis depends, of course, upon the exciting cause and upon the general condition of the patient before and during the disease; but it also hinges largely upon two factors:

1. The amount of urine secreted by the diseased kidneys.

2. The readiness with which the other avenues of elimination—*i.e.,* the breath, the sweat, the feces—can be made temporarily to take the place of the failing kidneys.

Treatment. Under this heading will be considered merely the treatment of the attack of acute nephritis— the after treatment, diet, etc., being taken up under chronic parenchymatous nephritis.

We have no drugs or other means at our command by which to cure acute nephritis. Since we cannot treat the disease, we must limit our efforts to treating the patient that has the disease.

The objects of treatment are two-fold:

1. To stimulate the kidneys to resume the secretion of urine.

2. To secure elimination of poisons by the other channels of excretion. As we cannot increase elimination from the lungs, treatment is directed towards:

     (a) The skin.

     (b) The bowels.

General Management. The patient should be in bed, in a well-ventilated room, protected from draughts, and at a temperature not below 70°, so that the skin will not be dried and its pores contracted by chilliness.

As a general rule, it is wise not to try to give any food for the first twenty-four hours. The patient is usually nauseated, and, in addition, not one iota of additional strain must be thrown upon the kidneys. Water, also, should be given in moderation. If forced upon the patient, it throws an added strain upon the kidneys. Acute nephritis, in its intense stage, is not a condition in which the kidneys need "flushing out." The thirst of the patient is a good guide as to the amount of water to be given. It is well to give water in small amounts, and rather frequently. When food in any form is considered advisable, milk is probably the best; cream can be added to it. Cereals are permissible, and fruits in moderation

are not injurious. During the acute stage of nephritis it is far safer to underfeed than to overfeed. What foods are given should consist almost entirely of fats and car- bohydrates, in order to call upon the kidneys as little as possible for the elimination of proteins.

Stimulation of the Kidneys to Secretion of Urine. Diuretics are here indicated. At first the stomach will hardly tolerate any drugs, but as soon as possible the physician in charge will order that diuretic in which he has the greatest faith. Lemonade is of service, a dram of cream of tartar being added to every pint of lemonade to increase the diuretic effect. The citrate and acetate of potassium and theobromin sodium salicylate (diuretin) or the infusion of digitalis and syrup of squills are some of the preparations most in use.

Stimulation of the Other Avenues of Elimination. This is brought about by:

(1) Catharsis—purgation.

(2) Diaphoresis—sweating.

1. *Catharsis.* It must be borne in mind that when catharsis is employed in as serious a condition as acute nephritis the object is not only to empty the bowels, but far more to abstract fluid from the body in order to lessen edema and eliminate poisons. To obtain this effect, many copious watery stools must be obtained. The drugs mainly relied upon are the salines, especially magnesium sulphate, which is frequently given in dram doses of a saturated solution every half-hour until eight or ten free, watery movements are obtained. Many pre- fer this method to the giving of one very large dose, as the stomach will often not tolerate the single dose. If needful, the more drastic purgatives, such as elaterium and croton oil, may be given.

2. *Diaphoresis.* Here, too, it must be emphasized that what is desired is not a gentle perspiration, but a profuse, dripping sweat. There are several methods of obtaining this:

    (1) Hot baths—hardly sufficient.
    (2) Wet hot pack.
    (3) Dry hot pack.
    (4) Pilocarpine hypodermically.

The wet and dry hot packs are the means mainly relied upon to obtain the desired effect. The packs are given from two to four times daily for from twenty minutes to an hour each, depending upon the patient's condition. During the pack an ice-cap should be applied to the head. The drenching sweat abstracts a large amount of fluid from the body, and is of great benefit. Pilocarpine is rarely used as the sole sweating agent, as once the sweat is started by the drug in full dosage it cannot be controlled, and may prove too exhausting. A small dose of pilocarpine is frequently given to the patient when in the hot pack, in order to start the sweat.

The pain over the kidneys in acute nephritis is often aided by the application of the hot-water bottle or a mustard plaster. Severe headache occasionally requires sedatives, sometimes necessitating morphia, which of course is always used sparingly, though at times it seems to increase the urinary flow.

When caring for a case of acute nephritis the nurse must secure, measure, and record with the greatest care the total amount of urine, as this is the most important factor concerning which the physician will desire information. She must also be keenly alive to the amount of edema present, its increase or decrease, and any change in the patient that may be suggestive of an approaching

uræmic convulsion. Acute nephritis in its intense forms
is a fierce fight; and there are few acute conditions in
which the chances for recovery depend so much upon
the mode of treatment instituted, and upon the care and
faithfulness with which the details of this treatment are
carried out.

CHAPTER XIII.

CHRONIC PARENCHYMATOUS NEPHRITIS.

CHRONIC parenchymatous nephritis differs from acute nephritis in degree only. The process in the kidney is one and the same, the causative factors are identical, and, generally speaking, the symptoms are also similar, save that they are less marked in the chronic than in the acute form. It is impossible to draw an absolute dividing line between a severe case of chronic parenchymatous nephritis and a comparatively mild case of acute nephritis.

It follows that a recital of the symptoms must largely be a repetition of those cited under acute nephritis.

Edema is a very constant symptom, and at times is very marked. Instead of being universal, it is noticeable mainly in the face in the morning, and in the legs in the evening, if the patient is up and about. It may become the chief complaint, going on to general anasarca, the patient finally dying "waterlogged."

The urinary changes are similar to those found in acute nephritis, save to a lesser degree. The urine is lessened in amount, of a rather high specific gravity (1.020 to 1.025), containing a large amount of albumin and many granular and hyaline casts.

Uræmic symptoms of varying intensity are almost always present. Headache, mental and physical inertia, nausea and vomiting are very common. There may be a profuse diarrhea. Anemia is generally present, often to a marked degree, and despite the increase in actual weight (due to edema) the patient looks, and is, emaciated. The heart is usually enlarged, and there is al-

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most always dyspnea, partly from heart-strain and partly because of the amount of edema. The symptoms of acute uræmia (*q.v.*) may occur at any time, but are not as apt to show themselves as in chronic interstitial nephritis.

The prognosis is very grave. Recovery is practically unknown. The kidneys are permanently damaged. The disease runs a course lasting from six months to three years, and is characterized by exacerbations, during which more and more kidney structure is damaged, and remissions during which some repair seems to take place. Death occurs either from the enormous edema, with fluid in the peritoneal cavity (ascites), in the pleural cavity (hydrothorax), and finally in the lungs (pulmonary edema), from uræmia, or from some intercurrent disease to which the patient, because of lowered resistance, is particularly susceptible.

Treatment. The principles of treatment are the same as those laid down for acute nephritis, *i.e.,* to spare the kidneys all possible strain, and to stimulate excretion through the other avenues of elimination. Heroic measures, however, are not usually necessary.

General Management. The patient may or may not be confined to bed, depending upon the general strength, the amount of edema, and particularly upon the comfort of the individual. If a patient is more at ease in an armchair than in bed, he is usually allowed to exercise his choice. Care should be taken to protect the patient from draughts, from cold, and especially from dampness. Great attention to the skin is necessary. Its nutrition is frequently markedly interfered with because of the edema, and it is very susceptible to infection. Frequent warm baths are essential, followed by an alcohol rub and

liberal powdering with talcum powder. If the patient is in bed, the position must frequently be changed, and pressure at once removed from any red or painful area of the skin.

Elimination through the bowels will, of course, be seen to by the attending physician, but the nurse must be very careful to call his attention to any signs of constipation, either as regards the number or character of the movements. Generally speaking, it is far better for the patient to have two or even three stools daily than to go a day without a thorough bowel evacuation. The amount of catharsis necessary will, of course, be measured by the general condition of the patient.

Diuretics are almost always employed, and have a great field of usefulness in these cases. Whether requested or not, the nurse should always keep a charted record of the total twenty-four-hour urine, so that it can at once be turned to for reference. A hot pack or two per week often proves of benefit, both in aiding elimination through the skin and in keeping it in good condition.

Diet. Formerly, patients with chronic parenchymatous nephritis were very markedly restricted as to their diet, especially as to variety, and were fed milk! milk!! milk!!! until their very lives became a burden. More recently, however, it has been found that these patients do quite as well on a more liberal allowance. The patient's proteid intake must be restricted—how much is to be determined in the individual case, but as a rule not over 80 to 100 Gm. are to be taken in twenty-four hours. Broadly speaking, the fats and carbohydrates can be freely taken, comprising, among other things, well-cooked green vegetables, root vegetables, cereals, fruits and simple desserts. Sugar may be given freely. Spices,

condiments, alcohol, meat soups and heavy meats are to be avoided.   It has been found, however, that a lamb chop or two, or a small piece of steak twice a week, do no harm, and materially help the patient's appetite and general morale.

Restriction of Fluids.   This depends largely upon the amount of edema.   If there is much dropsy, it is evident that water does not pass freely through the kidneys, and in such cases the amount of fluid intake is limited. If edema is very slight, or absent, it is frequently customary to give the patient enough water to bring the total amount of urine in twenty-four hours up to about 3 pints, or 50 ounces.   Water is one of the best diuretics, and in suitable cases is invaluable.

Salt Restriction.   In many cases of chronic parenchymatous nephritis the kidneys do not excrete salt (sodium chloride) satisfactorily.   Almost everyone eats much more salt with food than is necessary for use in the body, and the excess must be eliminated through the kidneys. In cases of nephritis with edema, many have adopted the rule to reduce the salt intake to 2 Gm. (30 grains) in twenty-four hours.   This is often at first a considerable hardship to the patient, but it is frequently necessary to enforce it in order to get rid of the edema.

Further Measures.   If ascites or hydrothorax are present, the fluid is generally withdrawn by tapping.   Insomnia must receive appropriate treatment by means of hypnotics and sedatives.   Iron in some form is usually given for the anemia.   To the highest degree must the treatment of chronic parenchymatous nephritis be one of individualization; the conscientious nurse, with a knowledge of what is being attempted, and why it is being tried, will be of inestimable help to both physician and patient.

CHAPTER XIV.

CHRONIC INTERSTITIAL NEPHRITIS AND ARTERIOSCLEROSIS.

(CARDIOVASCULAR-RENAL DISEASE.)

WHILE in textbooks on medicine chronic interstitial nephritis and arteriosclerosis are dealt with in separate chapters, for the sake of simplification it has seemed best to consider them together, and in that connection to dwell also upon the relationship borne and the part played by the heart, this triple combination being recognized and spoken of as cardiovascular-renal disease.

This disease is of very frequent occurrence, and is in fact one of the most common conditions seen in elderly persons. It gives rise to a great number of symptoms, but usually one of the elements (the heart, the blood-vessels, or the kidneys) is more prominent than either of the other two, and the majority of symptoms complained of or observed can be laid at the door of one of the three units going to make up the pathological condition.

Chronic interstitial nephritis and arteriosclerosis are so closely interwoven, and their causative factors so similar, that one is hardly ever seen without the other, and the almost invariable rise in blood-pressure at once throws an added strain on the heart, to which that organ, though it may bear up for a time, must in the end assuredly succumb.

The causes leading up to chronic interstitial nephritis and arteriosclerosis are:

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1. Age.   Over 55 years every individual undergoes changes presently to be described, which to a greater or lesser degree affect the kidneys and the arteries.

2. Alcohol, especially when used regularly over a long period of years, even if never to excess.

3. Over-eating, especially when this is combined, as it so often is, with a sedentary life, an overplus of alcohol and tobacco, too great a dependence upon the luxuries of life, and insufficient exercise.

4. Faulty metabolism, such as gout and diabetes.

5. Syphilis.

Pathology.   The essential feature in bringing about a condition of chronic interstitial nephritis and arteriosclerosis is the establishment of a process of fibrosis in the human body.   By fibrosis is meant the gradual transformation of normal tissue into dense, firm, inelastic fibrous tissue.   This process attacks mainly the kidneys and the arteries.   It is one of the signs of advancing years, one of the signals that the human machine has seen its best days, has done its best work, and is wearing out.   Many cases of general fibrosis occur simply as a result of age, none of the other causative factors playing any rôle whatsoever.

The kidneys are small, dark, mottled, and the capsule, instead of peeling off easily, is everywhere densely adherent, and when stripped brings away with it small pieces of kidney tissue.

Microscopic evidences of fibrous tissue formation while visible in the uriniferous tubules, are most marked in the glomeruli and in the interstitial tissue.   The glomeruli are largely destroyed, being in many instances reduced to mere fibrous tufts with no traces of capillaries, epithelium, or capsule of Bowman left.   The interstitial

tissue appears greatly increased in amount, the uriniferous tubules are relatively few, and the cells lining them are flattened out and show evidences of degeneration.

The arteries suffer most in their middle coat (the media). It is here that normally the elastic fibres are situated that allow the artery to expand with each beat of the pulse, and that, more important still, by their recoil help the heart to such a great extent to force the blood-current along. These elastic fibres are transformed to a greater or less extent (according to the severity of the process) into fibrous tissue, so that the vessel wall becomes comparatively rigid. In addition, deposits of lime salts occur in the arterial walls, sometimes to such an extent that the radial artery, when rolled under the finger, feels like a pipe stem.

It will readily be seen that with unyielding, inelastic arteries blood-pressure must rise, and with heightened blood-pressure the heart has to pump against an abnormally great resistance. This results in increased work on the part of the heart, showing itself by hypertrophy of that organ, especially of the left ventricle. In time, however, the heart shows signs of failing, and symptoms of cardiac dilatation make their appearance.

Symptoms. 1. *Urinary*. The amount of urine is greatly increased, and one of the first symptoms noted by the patient (and rarely complained of) is the necessity of arising two or three times during the night to empty the bladder. The total urine voided in twenty-four hours is usually from $2\frac{1}{2}$ to $3\frac{1}{2}$ quarts ($1\frac{1}{3}$ quarts being approximately normal). The specific gravity is very low, usually under 1.010. There may or may not be albumin present, and if found it is usually very slight in amount. On microscopical examination a few hyaline casts may

be seen. It is impossible in these cases to tell from an ordinary urinary examination the extent of damage to the kidney, and recourse must be had to the tests for kidney function (*q.v.*).

2. *Toxic.* Signs of uræmia may, and usually do, show themselves. It is not uncommon for patients with cardiovascular-renal disease to suddenly suffer from an attack of acute uræmia, with convulsions, suppression of urine, etc. More frequently, however, are seen the signs of chronic uræmia, which are an indication that the kidneys are eliminating insufficiently, and that poisons in the shape of waste products are being gradually accumulated within the system. These symptoms are: headache (especially in the morning), a coated tongue, some constipation, slight drowsiness, dizziness, mental depression, and a slight mental dullness.

3. *Cardiac.* The patient frequently suffers from palpitation, and dyspnea is very common, especially on slight exertion. Some cough is frequent and a chronic bronchitis, accompanied by much "wheezing," often is present as a result of heart weakness.

Sudden edema of the lungs may occur. Hemorrhages, especially nosebleed and blood in the urine, are frequent as a result of the giving way of small vessels because of the heightened blood-pressure. Apoplexy is a frequent occurrence, due to the rupture of a blood-vessel in the brain, because of the combination of inelastic walls and heightened blood-pressure.

Gastric and digestive disturbances are very common, loss of appetite, nausea, vomiting and constipation being most prominent.

Edema is absent save if an attack of acute nephritis occurs, which is not infrequently the case. The blood-

pressure is almost invariably raised, in some cases to alarming heights.

It will be seen that the symptoms of this disease of triple origin are so numerous that an accurate picture of the condition is very difficult to obtain, because of the number of elements involved. Probably no case will exhibit *all* the symptoms, and, as previously mentioned, those symptoms referable to one of the three elements at fault will usually dominate the scene.

Prognosis. The outlook is bad, as far as recovery is concerned. Permanent tissue changes have taken place which cannot be cured, and while by careful attention to hygiene, diet, and special symptoms, the patient's life can be considerably prolonged, and made in many instances very comfortable, yet in the end the fight is bound to be a losing one.

Treatment. The objects of treatment are threefold :
1. To spare the kidneys as much as possible.
2. To spare the heart as much as possible by reducing blood-pressure.
3. To relieve symptoms as they arise.

1. In attempting to spare the kidneys, the diet is essentially the same as that in chronic parenchymatous nephritis, save that reduction of salt is not usually necessary, for edema is rare, and when it appears, is usually of cardiac origin. Proteid must be given sparingly, not more than 80 to 100 Gm. in twenty-four hours.

Elimination through the bowels and skin is important, the latter being often well secured by a course of sweats every few weeks.

2. In the reduction of blood-pressure sweats work well, as abnormal tension is due, in part at least, to the kidneys. In addition, drugs are given whose action is to

lower blood-pressure. These drugs are known as vaso-dilators, because they cause the blood-vessels to dilate or expand, and therefore allow the contained blood to be under less pressure.

The main drugs used are:

(a) Amyl nitrite.
(b) Nitroglycerin.
(c) Sodium nitrite.

Of these, amyl nitrite acts most rapidly, but its effect is violent, often very uncomfortable to the patient, and very transient in duration. Nitroglycerin acts rapidly, but must be frequently repeated, as its action is also transient, and, when given in large doses, is apt to cause severe headache. Sodium nitrite acts more slowly than the two preceding, but its action is far more sustained, and it does not give rise to the disagreeable symptoms caused by the other two drugs. In addition, potassium iodide is very frequently given, though it has no power to reduce blood-pressure. The dosage and frequency of administration of these drugs will, of course, be determined by the attending physician.

3. The relief of individual symptoms has nothing to do with the actual treatment of the condition under consideration. These symptoms are so many and varied that a host of methods are in use, each physician having his preference. Sedatives of one kind or another will usually have to be employed, and in the vast majority of cases morphia will finally be resorted to, especially for the nocturnal dyspnea, which is often so distressing. During the earlier stages of the disease the patient should not be kept in bed, but encouraged to take mild exercise, always carefully abstaining from overfatigue. The care of the skin and bowels is all-important. Early and mod-

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erately advanced cases can usually do work, if that work is not manual and violent. Such cases will not need a nurse. The nurse sees these unfortunates when their days of activity are behind them, and when the exhausted and rapidly failing vital organs can no longer cope with the work demanded of them.

CHAPTER XV.

TESTS OF KIDNEY EFFICIENCY.
(RENAL FUNCTION TESTS.)

WITHIN the last ten years much light has been shed
upon the workings of the organs of our bodies, and
methods have been devised to test their working powers
or functional efficiency. In no organ has this power been
the subject of more investigation than in the kidney, and
in the case of no other organ have the results been as
satisfactory and as easily practicable for everyday use.
Fifteen years ago, if on a careful urinary examination the
specific gravity was normal, and no albumin, sugar, or
casts were found, it was taken for granted that the kid-
neys were in normal condition. Today the situation has
been complicated by the knowledge that seriously dis-
eased kidneys may secrete a urine by no means propor-
tionately abnormal. This insufficiency of excretion on
the part of the kidneys has been made plain by the dis-
covery of the renal function tests.

The nurse, of course, will not be called upon to make
these tests; neither is it necessary that she should know
their theoretical foundations.

The nurse will, however, assuredly care for many pa-
tients in whom these tests will be made, and she should,
in a general way, appreciate their significance and under-
stand their mode of application in order to more intelli-
gently co-operate with the physician in his work. There
are many tests for estimating kidney function, no one of
which, alone, can give a complete picture of the working

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powers of the kidney. Several of these tests are very complicated, require a laboratory expert for their performance, and can never become a routine in general practice. There are, however, two tests that can be performed with great ease, and that give very valuable information as to the functionating power of the kidneys.

These two tests are the ones most used by physicians in their practice, and the nurse should be familiar with the modes of procedure.

These tests are:

(1) Phenolsulphonephthalein test.

(2) Specific gravity test.

1. *Phenolsulphonephthalein test* (commonly known as the phthalein test).

This test is based upon the ability of the kidney to excrete a certain amount of this particular dye in a given length of time. The technique of performing the test is very simple. There are many slight modifications, the following being sufficiently accurate for general clinical work.

Fifteen minutes before making the test the patient is given 400 c.c. of water to drink. Immediately before beginning the test the bladder is emptied. One c.c. of a 1 per cent. solution of phenolsulphonephthalein (a brilliant red dye that is put up in ampoules specially for this test) is then injected into the muscles of the back. The patient is given no food or drink, and exactly *one hour and ten minutes* after receiving the injection empties the bladder, the entire amount of urine voided being placed in a bottle labeled thus: "1st hour." In exactly *one hour* more the bladder is again emptied, and the urine voided is placed in a bottle labeled: "2d hour." The

test, as far as the patient is concerned, is then at an end.

The urine in the bottle labeled "1st hour" is poured into a 1000-c.c. graduate, and, irrespective of its amount, water is added up to 1000 c.c. A few drops of 40 per cent. sodium hydrate are added, which causes the diluted urine to become pink or red, according to the amount of the dye present. A small portion of the contents of the graduate is then taken and compared with a standard scale in an instrument known as a colorimeter, the counterpart of the color of the urine being found on the scale, and the percentage of the dye excreted, read off, and recorded. The contents of the bottle labeled "2d hour" are similarly dealt with, and the percentage of the dye excreted during the first and second hours are added together.

Normal kidneys will excrete from 60 to 80 per cent. of the dye within two hours. Any percentage below 50 denotes that the kidneys are under-functioning, and when percentages of 35 and less are reached the patient is in danger of an attack of uræmia, even though there may be no symptoms of that condition present.

2. *Specific Gravity Test.* This very simple procedure shows the specific gravity of the urine at various hours during the day, demonstrates the presence or absence of nocturnal polyuria, and sets forth the fact whether the specific gravity of night urine is higher than that of day urine (normal) or whether it is the same (usually abnormal). The nurse can carry out this test for the physician with the greatest ease, the only accessories required being a 500 c.c. graduate and a specific gravity float (urinometer).

In cases where very great accuracy is desired, a defi-

nite "renal test-diet" is given. This diet has been carefully worked out by Dr. H. O. Mosenthal at the Johns Hopkins Hospital. When this is used, the exact amount of salt given is measured, and that not used is deducted from the total. For ordinary clinical purposes such accuracy is not essential, as is shown by the following quotation from one of Dr. Mosenthal's articles:

"In private practice it would only be necessary to ask the patient to eat three full meals a day, and write down the approximate quantities (as: 1 cup of coffee, 2 slices of toast, 2 tablespoonfuls of oatmeal, etc.), in order to be certain that the diet for the day contained a sufficient quantity of the diuretic materials of ordinary food to make an adequate demand on the kidney to test renal function.

"It is extremely desirable to insist that, since the food as found in most households suffices to carry out these tests, and the procedure is not a complicated one, it need not be confined to hospitals and patients who can afford private nurses."

Technique of the Test. No food or fluid to be given between meals, and none between 8 P.M. and 8 A.M. Patient empties bladder every two hours—at 8 A.M., 10 A.M., 12 noon, 2 P.M., 4 P.M., 6 P.M., and 8 P.M. If possible, patient is to go from 8 P.M. to 8 A.M. without voiding. If this is not possible, all urine voided between 8 P.M. and 8 A.M. is to be collected and mixed before taking specific gravity. Every voiding during the day is to be measured, and the specific gravity taken, and both findings recorded.

The following table is taken from Dr. Mosenthal's article and represents the findings in a normal individual:

Time of day	Urine c.c.	Sp. G.
8 to 10	153	1.016
10 to 12	156	1.019
12 to 2	194	1.012
2 to 4	260	1.014
4 to 6	114	1.020
6 to 8	238	1.010
Total day	1115	.....
Night, 8 to 8	375	1.020
Total, 24 hours	1490	.....

To be noted are: The variations occurring in the fluid output and in the specific gravity, which, generally speaking, are in inverse ratio, *i.e.,* the greater the amount of urine the lower the specific gravity; the small amount of night urine with high specific gravity.

In contrast to the preceding, the following table is shown, taken from an advanced case of chronic interstitial nephritis:

Time of day	Urine c.c.	Sp. G.
8 to 10	24	1.005
10 to 12	106	1.006
12 to 2	82	1.007
2 to 4	83	1.008
4 to 6	0	.....
6 to 8	230	1.008
Total day	525	.....
Night	1140	1.007
Total, 24 hours	1665	.....

To be noted are: the low specific gravity, with but very little variation (so-called "fixed" specific gravity), the small amount of day urine, and the large amount of night urine with a low specific gravity.

This test is very valuable, and, taken in conjunction with the phthalein test, gives a practical working idea of the functional ability of the kidneys. By means of the routine employment of these tests improvement or the reverse can be closely followed, the possibility of serious developments such as acute uræmia foreshadowed, and thus nurse and physician kept on their guard.

CHAPTER XVI.

CEREBRAL HEMORRHAGE (APOPLEXY) AND CEREBRAL THROMBOSIS.

Nature of the Conditions. By cerebral hemorrhage is meant the escape of blood into the tissue of the brain by the bursting of one of its blood-vessels.

By cerebral thrombosis is meant the stopping up of one or more of the vessels of the brain by a clot or thrombus, thus depriving of its blood-supply the area of brain nourished by the occluded vessel.

These conditions occur usually in middle life or in old age, and have as their one great cause, arteriosclerosis. High blood-pressure, coupled with inelastic, unyielding vessel walls, predispose to hemorrhage, while low blood-pressure and the roughened interior of arteriosclerotic vessels result frequently in the formation of thrombi or clots.

Cerebral hemorrhage and thrombosis may occur in the comparatively young (30 to 40) if the arteries are sclerotic, and this is especially apt to be the case in syphilitic subjects.

Cerebral Hemorrhage.

Symptoms. The symptoms vary greatly in degree, and depend upon the location and the size of the hemorrhage. If the hemorrhage is very large, the patient may drop dead. In less severe cases the "stroke" may cause unconsciousness, persisting for several days, accompanied by stertorous breathing, slow pulse, and a hemi-

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plegia, *i.e.*, complete paralysis of one-half the body, extending from head to heel. The paralysis always occurs on the opposite side from the hemorrhage, due to the crossing of the nerve fibres from one side of the brain to the other. The face is paralyzed, the smile is crooked, the corner of the mouth being drawn over toward the sound side. The tongue when protruded points away from the side of paralysis. The arm, hand, thigh, leg, and foot on one side are incapable of any motion, and may also feel numb, or else be notably insensitive to touch. Depending upon the site of the hemorrhage, there may be various disturbances of speech, known as aphasia, the patient being unable to utter the word desired, unable to remember words, using the wrong word, *e.g.*, saying "hat" for "breakfast," "shoe" for "toothbrush," etc., thus being wholly unintelligible. The functions of rectum and bladder may be interfered with, constipation being present or else both urine and feces being voided involuntarily.

In milder cases the paralysis may be more localized, one arm, one leg, or the face only being affected, and at the time of the "stroke" there may be no unconsciousness.

The course of the disease is subject to great variations. Complete recovery is not to be expected in a hemorrhage of any size, as a certain portion of braintissue is always permanently destroyed. Usually there is some improvement for a while after the "stroke," as portions of the brain affected slightly by the hemorrhage, but not destroyed, regain and resume their function. A period eventually is reached where no further improvement takes place, and the patient continues for an indefinite period in his paralyzed condition. Death occurs

from successive "strokes" or hemorrhages, from exhaustion, from infection of bladder and kidneys, or from bed-sores, which are very apt to occur, as the nutrition of the skin is often interfered with.

Treatment. The patient suffering from an apoplectic stroke is to be put to bed, with the head high and the feet low. One of the objects of treatment is to draw as much blood as possible *away* from the brain. For this purpose an ice-bag may be placed on the head, and heat applied to the feet. Free catharsis is desirable, in order to abstract blood and to lower blood-pressure. To obtain many bowel movements in an unconscious patient croton oil is often used. Drugs to quiet the heart's action and reduce blood-pressure, such as aconite and veratrum viride, are frequently given. An ice-bag over the heart is often effective. Some cases are benefited by bleeding 12 to 16 ounces from a vein (venesection).

The nurse must be careful to frequently change the patient's position in order to lessen the likelihood of hypostatic pneumonia, as well as to lessen continued pressure upon any one spot on the skin. She must also pay particular attention to keeping the skin clean and dry (which is often difficult because of the involuntary movements), as bed-sores are apt to develop, and, once present, to spread rapidly, and sometimes cause the death of the patient. The nurse must also report to the physician whether a sufficient amount of urine is being voided, as these patients often allow the bladder to become overdistended, and only the "overflow" of urine is passed.

For the frequent restlessness, especially at night, sedatives are necessary, such as the bromides and chloral, and morphia must often be resorted to.

Cerebral Thrombosis.

The symptoms of cerebral thrombosis are neither as marked nor as striking as those of cerebral hemorrhage, though, in the end, the damage done may be fully as great. If a vessel is gradually stopped up, the function of that part of the brain which it supplies is, of course, gradually interfered with. The symptoms depend entirely upon the location of the thrombosis. There may be weakness, numbness, or a sense of "pins and needles" in a hand or arm; dizziness; thick, difficult speech, and any of the different forms of aphasia. Loss of consciousness is not the rule, though it may occur if the occluded vessel is very large. The thrombotic process usually spreads, so that eventually hemiplegia may result.

As in the case of apoplexy, perfect recovery cannot occur, as some portion of the brain is permanently damaged. If the process does not spread, certain parts of the affected brain-tissue may regain their function by receiving blood from other vessels than the one that has become occluded (collateral circulation), but a certain amount of damage is irreparable.

Treatment. As these cases are usually associated with low blood-pressure, the object of treatment is the opposite of that advocated for cerebral hemorrhage, *i.e.,* it is desired to drive blood *to* the brain. The head should be low and the feet elevated. Heat over the heart is advisable, and profuse catharsis is usually not practised. Stimulation is often indicated, and, in addition, drugs to prevent constriction of the cerebral blood-vessels, of which nitroglycerin is the best example. The remainder of the management of the patient differs in no way from that outlined under cerebral hemorrhage.

CHAPTER XVII.

PLEURISY.
(Dry and With Effusion.)

By pleurisy is meant inflammation of the pleura, which is the serous membrane that surrounds the lung.

The pleura is formed of two layers, one lying directly against the lungs (the visceral pleura), the other lying between the visceral pleura and the inner surface of the
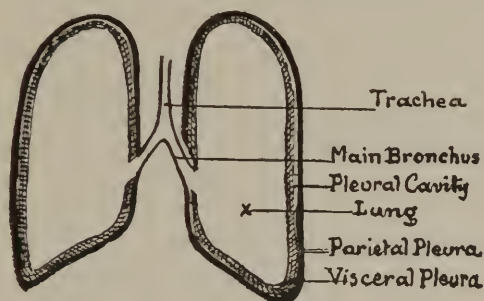
Fig. 4.—Diagram of pleural sacs.

ribs, and known as the parietal pleura. These two layers of pleura become continuous with each other, above and below the hilus or root of the lung. (See diagram.)

The parietal and visceral layers of pleura are constantly in contact, a few drops of fluid being found between them for purposes of lubrication. The so-called "pleural cavity" lies between the parietal and visceral layers. In health there is no "space" or "cavity," the two layers being closely applied one against the other. Both pleural sacs are wholly and entirely separate and distinct, one from the other, there being one for each lung.

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It is often very difficult for nurses to appreciate what is meant by the "pleural cavity." The following simile may be of assistance.

If the stopper is removed from a hot-water bottle, if the bottle is then rolled up so as to drive out all the air, and if then the stopper is tightly screwed in, it will be impossible to separate the two layers of rubber forming the hot-water bottle, because of the pressure of air from the outside. Yet it would be easy enough to figure to one's self a "cavity" within the hot-water bottle, if only air or water could be gotten in between the two layers. It is in the same sense that the pleural "cavity" exists. Normally the layers are as close to each other as the sides of the hot-water bottle, differing from it in that they slide against each other with every respiratory movement of the lungs. If fluid or air appears between the layers of the pleura they are forced apart and the "pleural cavity," from being a feat of the imagination, becomes an accomplished fact.

It is important to understand this question of the pleural cavity in order to appreciate conditions in pleurisy with effusion, and in pneumothorax, whether spontaneous or artificial. (See last section in chapter on Tuberculosis.)

There are two varieties of pleurisy:

(1) Dry pleurisy.

(2) Pleurisy with effusion.

1. **Dry Pleurisy.** In dry pleurisy there is congestion of the two layers of the membrane over the site of inflammation, and minute threads of fibrin extend from one layer of the pleura to the other. When the act of respiration takes place, instead of gliding smoothly over each other, the two pleural surfaces grate, the fibrin

strands stretch and break, and, as the pleura is very richly supplied with nerves, intense pain is felt over the inflamed area.

Etiological factors.

    (1) Cold, especially in connection with getting wet.
    (2) Trauma, *c.g.,* a blow or a kick.
    (3) Infectious diseases, especially:
        (*a*) Lobar pneumonia (dry pleurisy always present).
        (*b*) Tuberculosis; 90 per cent. of idiopathic cases of pleurisy are of tuberculous origin.

Symptoms.

Pain. Sharp, knife-like, lancinating. May be localized or spread over quite a wide area. Usually most marked at the side of the chest and toward the base of the lung. The pain is increased by deep breathing, and is often severe enough to "cut off the breath." It is also increased by laughing, sneezing, coughing, blowing the nose, or any inspiratory or expiratory effort.

Dyspnea. Due to the pain, which necessitates frequent short breaths. Sometimes the dyspnea is so intense as to produce a slight grade of cyanosis. It has often been noticed that a hypodermic of morphia, by relieving the pain and permitting deeper breaths, will wholly dissipate the cyanosis.

Fever. Usually low—under 101°. Many cases show no fever. Some have as much as 102°.

Treatment.

Drugs. If the pain is bad, codeine, either by mouth or by hypodermic, usually gives relief, though not infrequently morphia is necessary. Some patients are benefited by acetylsalicylic acid (aspirin).

Local Measures. Some form of counterirritation is almost always used. The means most employed are:

(1) Heat (hot-water bottle).

(2) Iodine.

(3) Mustard plaster.

(4) Antiphlogistine.

(5) Cantharides blister.

Strapping with adhesive plaster:

Strapping. Incorrect strapping is useless. The straps must be put on tight, and must remain tight if their action, to act as a splint and to limit motion, is to be obtained.

Adherence to the following simple suggestions will ensure tight and correct strapping:

(*a*) Use 2½-inch or 3-inch adhesive.

(*b*) Cut strips so that they will reach a little more than half way around the body, from a little beyond the sternum to a little beyond the spine. Unless the ends of the adhesive strips are firmly anchored to the "fixed" points of the thorax (the sternum and the spine) they cannot remain tight.

(*c*) Always have the patient sitting or standing when strapping is to be done, with hand on side to be strapped resting on top of head.

(*d*) If the chest is hairy, shave it.

(*e*) Tell the patient to exhale (empty the chest), and to hold it emptied during the application of each strap.

(*f*) Bring straps above and below the nipple in women, and in men protect nipple with a piece of cotton.

Straps should not be left in place more than four or five days, because of the irritation to the underlying skin.

2. **Pleurisy with Effusion.** At times an exudate is poured out from the surfaces of the pleural layers, and fluid appears in the "pleural cavity." The amount of this fluid varies greatly. There may be an ounce or so, or as much as three or four quarts. The fluid in typical cases is a clear straw color. It may, however, be turbid from the presence of pus, or it may be bloody. The latter condition occurs most frequently in malignant disease of the lungs and mediastinum.

Symptoms. Pleurisy with effusion is almost always preceded by dry pleurisy, with its characteristic symptoms. With the development of fluid there is:

1. *Cessation of pain,* due to the mechanical separation of the pleural layers by the accumulating fluid.

2. *Moderate fever*—101° to 103°.

3. *Increasing Dyspnea.* As more and more fluid collects, the lung on that side is markedly compressed, the heart is displaced to right or left (away from the fluid), as the case may be, and any physical exertion greatly increases dyspnea. The patient is often comfortable only in the erect posture. Cyanosis may set in.

4. *Sense of Weight.* A heaviness is complained of on the side where the fluid is to be found. As a chest two-thirds full of fluid contains about four or five pounds of water, this is not to be wondered at. Patients frequently complain of feeling the fluid "slosh" about whenever they change their position.

5. *Loss of appetite, nausea and vomiting;* especially in left-sided cases, because there is nothing between the fluid and the stomach but the left leaf of the diaphragm.

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The weight of the fluid constantly pressing on the stomach causes annoying symptoms.

Treatment.

Bed during the febrile period, light diet, and attention to the bowels. Many effusions need no interference, and, in time, become absorbed. Many physicians follow the rule not to interfere with a pleuritic effusion if it does not inconvenience the patient, as shown by fever, dyspnea and cyanosis.

If it is decided to try to remove the effusion, there are two methods of procedure:

1. To facilitate and hasten absorption by the giving of large doses of saline cathartics, which abstract fluid from the body. This method is not much in use at present.

2. Paracentesis, *i.e.*, tapping. The space selected is usually the seventh intercostal space, in either the posterior axillary or the mid-scapular lines. The patient is usually placed in a sitting posture, propped against pillows. If there is much fluid, the physician often finds it advisable to withdraw not more than 1000 c.c. at one time, as the change in pressure within the chest, or the change in position of the heart, may bring about unpleasant symptoms. Tapping in the vast majority of instances is a harmless procedure. At any sign of faintness, nausea, uncontrollable cough or profuse, frothy expectoration on the part of the patient, it is usual to stop the procedure. If only 1000 c.c. are drawn off, the remainder of the fluid may be aspirated in a few days.

Sometimes one tapping will suffice. Sometimes the fluid re-collects, and successive tappings are required. Latterly, some physicians have adopted the practice of removing the fluid and substituting nitrogen gas or air. (See section on Artificial Pneumothorax in chapter on Tuberculosis.)

LOBAR PNEUMONIA.

Definition. "An infectious disease characterized by inflammation of the lungs, toxæmia of varying intensity, and a fever that terminates abruptly by crisis" (Osler).

Etiology. The causative factor in lobar pneumonia is the pneumococcus of Fränkel and Weichselbaum, which was discovered in 1884.

Contributory causative factors:

1. Geographical. Pneumonia occurs in all climates, but is less frequent in the arctic and antarctic regions and near the equator, and most frequent in the temperate zones.

2. Season. The winter and spring months show the largest number of cases of pneumonia.

3. Cold. Not in itself enough to cause pneumonia. For instance, there were but few cases of pneumonia recorded during the celebrated retreat of the French army from Moscow in the dead of winter in 1812. Cold associated with getting wet is a very potent factor in bringing about the disease. Immersion in water, getting wet and chilled, and being unable to change one's clothes promptly, often determines the onset of pneumonia.

4. Injury to the chest, such as a blow or a kick, is unquestionably a factor at times in the causation of pneumonia.

5. Inhalation. Especially of ether, and after tracheotomy, as then the defensive mechanism of the upper air-passages is done away with.

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6. Alcoholism. A powerful factor in reducing bodily resistance, and predisposing to the disease. Alcoholism, wet, and exposure to cold, so often found in combination, furnish a fertile ground for the development of pneumonia.

7. Age. While occurring at all times of life, individuals between 20 and 30 are most prone to the disease. Thus, among 32,681 cases, 8,041, or 24.6 per cent., occurred in the third decade. Males are more frequently affected than are females, in the proportion of 6 to 4.

Pathology. There are four stages in the pathology of lobar pneumonia:

(1) Engorgement.
(2) Red hepatization.
(3) Grey hepatization.
(4) Resolution.

1. *Engorgement.* The pulmonary capillaries are markedly congested, and some red blood-cells appear in the air-spaces of the lung.

2. *Red Hepatization.* The affected lobe is in the process of consolidation or solidification. The air-spaces are more or less filled with an exudate composed of red blood-cells, fibrin, and some white blood-cells. The lung is much firmer than normal, is swollen, and pits on pressure.

3. *Grey Hepatization.* The affected lobe is solid. If a piece of it be put in water, it at once sinks, showing that it contains no air. The lung capillaries are obliterated, and the exudate is most abundant, being composed of some red blood-cells, but chiefly of white blood-cells, fibrinous threads, and bits of detritus. The lobe pits deeply on pressure, and is more or less of the consistency of liver.

4. *Resolution.* This is the beginning of the stage of repair. The fibrin threads break up. The white blood-cells show fatty degeneration. The lobe loses its solid feel, and becomes more soft and boggy. The capillaries again become visible, and gradually the entire exudate is completely absorbed, leaving the lobe in its former normal condition.

Symptoms. A short description of a typical attack of lobar pneumonia will first be given, and then the various symptoms will be taken up more in detail.

Typical Attack. An individual in apparently perfect health is suddenly seized with a hard, shaking chill, coupled with a sharp, intense pain in the side, and high fever, ranging from 103° to 105°. The face is flushed, the eyes bright, the expression anxious, the pulse full and bounding, the respirations rapid and shallow. There is cough, dry and hacking at first, later deeper and looser. The fever ranges between 102° and 105°, with slight morning remissions, until it terminates by crisis—that is to say, the fever drops several degrees within a few hours, and leaves the patient relatively comfortable. Herpes frequently appears on the lips. There may be cyanosis, and delirium is common at the height of the disease, which lasts on an average about a week. Convalescence is usually fairly rapid in those that recover, but the mortality is high.

Symptoms in Detail. Onset. There are two types of onset:

1. Sudden, described in the preceding paragraph. This is the more common mode in patients with good resisting powers.

2. Gradual. This mode of onset often denotes poor resistance. The temperature in this case does not run

high. The mental symptoms, especially stupor, are more marked, consolidation of the lung is protracted, complications are more to be feared, and mortality is higher. The gradual mode of onset occurs more frequently in individuals past middle life.

Pain. Caused by the inflammation of the pleura over the involved portion of the lung, resulting in a dry pleurisy. The pain is best described as lancinating, and is often referred to as a "stitch in the side." Sometimes when that portion of the pleura resting on the diaphragm is involved, the pain may be referred to the abdomen, and instances are on record where a diagnosis of appendicitis has been made. The pain is aggravated by talking, deep breathing, sneezing, coughing, and any act that causes increased friction between the inflamed layers of the pleural membrane.

Fever. This varies greatly. In typical cases, in patients with good resisting power, it usually runs from 103° to 105° at the height of the disease.

The crisis is sometimes preceded by a greater elevation of temperature. It occurs most frequently on the 5th, 7th, and 9th days of the disease.

The crisis in typical lobar pneumonia is one of the most striking occurrences met with in disease. A patient that has been running a temperature from 103° to 105°, that has been cyanotic, with labored breathing, with a pulse-rate between 120 and 130, perhaps actively delirious, and obviously in every way desperately ill, will, at the end of from six to eighteen hours, be found with a practically normal temperature, a respiration-rate but little above normal, the pulse-rate in proportion, all signs of delirium gone, a gentle perspiration taking the place of the raging fever, and the entire picture transformed

from one of extreme critical illness to one of relative comfort and comparative safety. *Moreover, on physical examination the lungs will be found to present almost identically the same signs that existed before the crisis.* What has happened? It would appear that the general infection has run its course, has finished its work, and, having fought to its last gasp, has suddenly unconditionally surrendered.
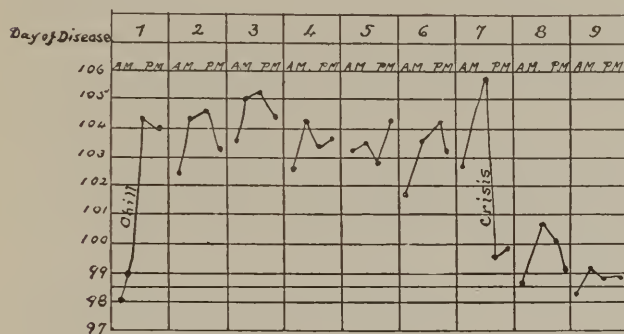
Fig. 5.—Temperature curve in typical attack of lobar pneumonia.

Often, indeed, it must be noted, the occurrence of the crisis is marked by very serious symptoms of heart and respiratory failure, necessitating great watchfulness and vigorous stimulation ; but if the patient can be safely tided over a few very anxious hours, the outlook is good.

The Pulse. At first the pulse is full and bounding. Its rate is usually about 120, though it may be somewhat faster. As the disease progresses the pulse becomes smaller and weaker. Sir James Mackenzie, a great English authority, sums up the question of the pulse in

lobar pneumonia so well in a single paragraph that I can do no better than to quote his exact words. He says:

"In all cases of acute lobar pneumonia that I have met, when the pulse showed even an occasional irregularity before the crisis was reached, death supervened. I have not found a single exception to this rule for over ten years, and while extended experience may prove it fallacious, irregularity of the pulse in pneumonia must, at all events, be looked upon as a most serious symptom. In pneumonia, the amount of arterial tension, the rate of the pulse and its rhythm are each of them among the most important indications we possess. Within a few hours after a rigor the fatal termination may be too plainly foretold by the character of the pulse. I have never seen an adult with a pulse-rate over 140 recover."

This excellent summary shows how important an indication is the pulse, and how fully the nurse caring for a patient with pneumonia must familiarize herself with the condition of the circulation as expressed in the pulse. The nurse rather than the doctor should be the first one to discover any abnormality or change in the pulse. Often and often a life will be saved by her watchfulness, observation, and timely warning.

The Heart. While the nurse will not examine the hearts of her pneumonia patients, and while the pulse is her index as to the condition of the heart itself, still she should know that the strain in pneumonia is thrown primarily on the heart, and that which is most dreaded is myocardial degeneration—that is, failure and exhaustion of the heart muscle. This weakness may arise from many causes. The three most commonly causing it are:

1. Toxemia. The general poison of the disease acts as a distinctly harmful agent upon the muscle fibres forming the heart.

2. Extensive pulmonary consolidation. When two or three lobes of lung are solidified, the heart may have such difficulty in pumping the blood through the relatively small unconsolidated space, that it gives way under the strain.

3. Hyperpyrexia. (Excessively high temperature.) When the temperature reaches extreme heights, over 106°, this condition in itself exerts a degenerative effect upon the heart-muscle.

Respiration. The respiration is rapid—30 to 50 per minute—short and shallow. There is often an expiratory grunt. The nostrils are seen to dilate markedly with each inspiration. Respiration is voluntarily and involuntarily restricted. Owing to the pain of the associated pleurisy respiration is shallow, and therefore carbon dioxide (CO_2) accumulates in the blood. This exerts a paralyzing effect upon the cells in the brain, where the respiratory centre is located, and this paralyzing effect still further hampers respiration. There is often some cyanosis, especially of the lips and finger tips, though in bad cases the entire face may take on a dusky hue.

Cough. Cough is an almost constant symptom in pneumonia. Jürgensen says: "It is rarely useful, always troublesome, sometimes dangerous." The cough is at first hard, dry, hacking, and paroxysmal. It is exquisitely painful, owing to pleurisy. Later it becomes looser, and abundant sputum is raised.

Sputum. The sputum at first is very characteristic of pneumonia. It is tinged with blood, this tinge giving

it a color best described as "rusty." It is extremely viscid and tenacious. The cup or glass into which it is expectorated can be turned upside down without the sputum being spilled. I have known the sputum to be so tenacious that it had to be wiped out of the patient's mouth and actually pulled off the tongue. As the disease progresses, and especially when the stage of resolution is reached, the sputum becomes less viscid, far more abundant, and often of a greenish color.

Herpes. This consists in the appearance on the lips and at the angles of the mouth of vesicles which dry up, leaving reddish-brown scabs. Herpes, while most common on the lips, may occur anywhere. Its presence is considered by some to be a favorable sign.

Urine. The main feature of the urine in pneumonia is a decrease in the chloride content, due to the large amount of chlorides contained in the exudate in the lungs. Save for this fact, the urine presents the usual characteristics found in most acute febrile diseases.

Bowels. There is usually constipation, though the bowels present nothing characteristic. In bad cases, there may be marked tympanites, which may prove dangerous by exerting upward pressure on the heart.

Blood. An increase in the white blood-cells is the rule (leukocytosis). The white blood-cells in pneumonia usually number from 15,000 to 50,000 (4000 to 7000 being the normal number). Leukocytosis is an important sign from the standpoint of diagnosis and prognosis. Patients with little or no leukocytosis almost always do badly.

Duration of Disease. Two days to three weeks; usually about ten days. The longer cases must be considered as suspicious. Complications may have set in.

Complications:

1. Pleurisy.

    (*a*) Dry. Practically a part of the disease.

    (*b*) With effusion. Develops in about 6 per cent. of the cases.

2. Empyema. Occurs in 2 per cent. to 5 per cent. of the cases.

3. Abscess and gangrene of the lung. These are rare.

4. Endocarditis.

    (*a*) Simple. Fairly common.

    (*b*) Malignant.

5. Pericarditis.

6. Acute nephritis. Not uncommon.

In alcoholic patients the mental and nervous symptoms predominate, delirium is violent and protracted, and the mortality is very high.

Prognosis. Always grave. Depends upon the following factors: Course of temperature, pulse, and respiration; age of the patient, pneumonia being most fatal at extremes of life; alcoholism, or its absence; altitude, pneumonia being more fatal at high altitudes than at low ones; the amount of lung involvement, and the occurrence of complications. According to Wells, the mortality in 465,400 cases was 94,826, or 20.4 per cent.

Treatment. We have at our command no specific for the treatment of lobar pneumonia. As we cannot, therefore, treat the *disease,* we must devote all of our efforts to treating the *patient.* The objects of treatment are threefold:

1. To facilitate the body's efforts in its own behalf, by means of:

(*a*) Rest.
(*b*) Hygiene.
(*c*) Diet.

2. To reinforce nature's proceedings along her own lines.

3. To support such organs as show signs of failing.

Every physician in treating pneumonia has, in all probability, a method that he prefers to all others, and the nurse will, of course, faithfully carry out his orders to the letter. The following suggestions for treatment are based on broad general principles, and, taken collectively, will be used in practically every case.

1. The patient must always be at *rest in bed* in the recumbent position, and the use of bed-pan and bed-urinal insisted upon.

2. *Fresh Air.* A patient with a serious infection and with markedly diminished breathing space obviously requires all the fresh air obtainable. In many hospitals pneumonia patients are being kept out of doors, with very gratifying results, both as regards the comfort of the patients and the percentage of mortality. In private homes this mode of treatment is seldom practicable. The room should be airy, and the windows kept open. This will necessitate added vigilance on the part of the nurse, in order not to allow the patient to become uncovered. Windows and doors should, of course, be closed in cold weather during the bath, the use of the bed-pan, examination, or any procedure involving exposure of the patient.

3. *Care of the Mouth, Tongue, and Teeth.* Toothpicks with cotton on the end, which are soaked in 2 per

cent. boric acid, are good for cleaning the teeth. A whalebone is an excellent instrument for scraping and cleaning the tongue; and sweet oil, cold cream, cocoa butter, or vaseline are welcome applications to lips excoriated and raw from herpes.

The condition of the mouth is a very good indication of the general care a patient is getting. The nurse that allows her patient's mouth to get in a foul condition is probably slighting that patient in other directions.

4. *Diet*. The diet should be liquid or semi-solid; small amounts frequently administered. If the patient is able to take semi-solid food, many physicians believe in giving it. The main articles of diet are chosen from the following list: milk, oatmeal, rice, hominy, eggs, cup custard, ice cream, broths, gelatine, jellies, and the various substitutes for milk.

Sufficient water should be given to slake the thirst. If the patient shows signs of becoming stuporous, it is well to force the amount of water.

5. *Bowels*. An initial purge with calomel, followed by a saline, is usually given. Later, the bowels are moved by enemata, though some physicians prefer laxatives by mouth.

6. *Fever*. High fever, save when accompanied by delirium or marked signs of toxemia, is generally not interfered with. With very high temperatures—106° or over—cold packs and sponges are resorted to. At the present time the majority of physicians do not look with favor upon the administration of drugs to reduce the fever of pneumonia patients.

7. *Cough*. When due to pleurisy, heat or cold, mustard, iodine, or strapping with adhesive plaster over the painful area will often help. Strapping has the disad-

vantage that it interferes with subsequent chest examinations. Often, drugs, such as codeine, heroin, or even morphia must be resorted to.

The productive cough that brings up sputum is, on the whole, beneficial, and is usually not treated. If very exhausting, steam inhalations will often be of aid. Some expectorant mixture is usually prescribed.

8. *Toxemia.* Usually best combated by forcing the patient to drink as much water as possible, and by injecting salt solution under the skin (hypodermoclysis), or by introducing salt solution into the bowel and allowing it to become absorbed (enteroclysis).

9. *Delirium.* This is helped by the methods mentioned in the preceding paragraph. The most efficient and most used drug for this condition is morphia, as in addition to quieting the patient it causes restful sleep.

10. *Circulatory System.* This must be most carefully watched by the nurse, and the slightest danger sign communicated to the physician. While each physician will, of course, order such circulatory stimulants as he sees fit, it may be wise to mention in passing the main drugs used for this purpose: caffein, camphor, adrenalin, digitalis, strophanthus, and strychnine. In severe cases, in full-blooded individuals with marked labored breathing and cyanosis, bleeding from a vein to the amount of 12 to 16 ounces is often of great help.

11. *Specific Treatment.* It has been found by Cole and his associates at the Rockefeller Institute that the pneumococci present in the sputum of pneumonia patients can be divided into types which have been designated I, II, III, IV. In any given case sputum is taken to the laboratory, and the type to which the pneumococci belong is determined. An immunizing serum corres-

ponding to the particular type is then administered. Good results have been obtained from this method of treatment, especially in cases showing pneumococci of type I.

12. *Collapse*. Collapse in pneumonia may occur at any time. Its onset is sudden, and the likelihood of its occurrence increases as the time of the crisis approaches. The main symptoms are:

Rapid prostration.

Chilly sensations.

Ashen face.

Cold, clammy skin.

Restlessness and air-hunger.

Rapid, shallow, gasping respiration.

Soft, compressible, often almost imperceptible pulse.

The condition is a most urgent one, and prompt action is necessary. The nurse may not be able to get the doctor at once, and her patient may die while she is calling up various numbers on the phone, trying to locate the physician. She must act on her own initiative. The following plan is set forth in the belief that it will meet with no opposition from any physician:

Apply heat to the extremities in the shape of hot cloths, hot bottles and hot-water bags.

Give diffusible cardiac stimulants:

(1) Strong ammonia on towel held near patient's nose;

(2) Aromatic spirits of ammonia, 1 teaspoonful in water;

(3) Camphor in oil, 3 to 6 grains hypodermically;

(4) Caffein, 2 to 5 grains hypodermically;

(5) Strychnine, $\frac{1}{30}$ to $\frac{1}{15}$ grain hypodermically;

(6) Adrenalin chloride, 1 : 1000 solution, 20 minims hypodermically;

(7) Ether, 15 to 20 drops hypodermically;

(8) Whiskey, 15 to 20 drops hypodermically.

The nurse should not be satisfied with giving any one of these alone. It will not be necessary to give them all, but two, three or four should be given, as in such a crisis the heart seems to react better from the effects of a "broadside" than from "single shots." It seems useless to add that the physician should be summoned at once, the nurse getting some person to trace him unceasingly until he is found.

13. *Convalescence.* It is quite impossible to give any absolute rules for the management of convalescence, for each case is a law unto itself. Convalescence in uncomplicated pneumonia is relatively rapid. Care must be exercised when the patient first sits up in bed, and on no account must he be allowed suddenly to raise himself, as several cases of sudden death from the unexpected strain on the heart are on record, as the result of such an indiscretion. Progress must be slowly made, and vigilance must never be relaxed until the nurse is dismissed from the case.

CHAPTER XIX.

BRONCHOPNEUMONIA.

BRONCHOPNEUMONIA is perhaps not so much a disease as a condition or lesion, and follows no set rules, either as to its causation or symptoms. It attacks preferably those at the extremes of life: infants, young children, and old persons. It rarely occcurs as a primary disease, but usually as a sequel of a pre-existing bronchitis, and is particularly frequent as a complication of various diseases, especially the acute eruptive fevers of childhood, such as measles. Bronchopneumonia frequently occurs after or during whooping-cough.

No specific organism is the causative factor in bronchopneumonia, though the disease is invariably of germ origin. The pneumococcus, the staphylococcus and the influenza bacillus are all found, the pneumococcus being present most frequently.

The pneumonic area, or area of consolidation, occurs in small patches, surrounding a bronchus, which is found filled with gray mucus, while the surrounding air-vesicles are filled with an exudate somewhat like that found in lobar pneumonia.

Symptoms.

1. *In Children.* Following a bronchitis, with its slight temperature, cough and expectoration (if the child is not too young to raise anything), bronchopneumonia sets in with increase in all these symptoms. The child becomes restless, and cough and dyspnea become more marked. Respiration is short, shallow, and may range from 50 to 75 per minute. Cyanosis may be present.

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The nostrils dilate with every inspiration, and the child is seen to strain to get its breath. Temperature varies greatly, usually reaching 103°, though it may rise as
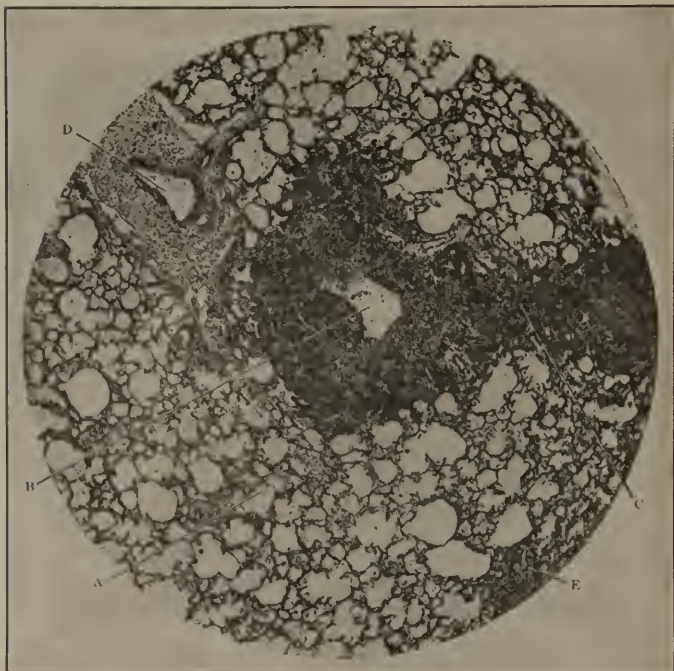
Fig. 6.—Bronchopneumonia.   A, normal lung tissue; B, small bronchus surrounded by consolidated lung tissue; C, thickened wall of bronchus; D, blood-vessels; E, small bronchus. ("Infancy and Childhood," Holt.)

high as 105°, and may reach 108° before death. The fever curve is irregular, but constantly above normal. The pulse is always rapid, often reaching 140 beats per minute. Sometimes it is so fast that it cannot be

counted. Vomiting and diarrhea are frequent. The temperature falls gradually, the entire duration of the disease being from two to three weeks. The child may improve, and then, with the formation of fresh pneumonic patches, all the symptoms may return with their former or with increased severity.

2. *In the Aged.* In old people there are frequently no definite symptoms, and the signs of bronchopneumonia are discovered by the physician during a routine examination of the lungs. Cough and sputum may be slight or absent, a low grade of temperature (100° to 101°) may or may not be present, and the main symptoms noted may be an increase in dyspnea on very slight exertion, and a gradual failure in strength. So many of these cases occur in elderly persons suffering from a chronic bronchitis of long standing, that the development of a bronchopneumonia is thought to be merely a flare-up of the bronchitis. The disease is very apt to attack persons that are bed-ridden from other causes, whose hearts are so weak that the blood is not propelled through the lungs with proper velocity, and who lie for days and weeks in almost the same position.

Prognosis. This depends largely on two factors:

1. Age. The younger the child the greater the mortality; the older the patient the greater the mortality. Probably from 30 to 50 per cent. of all cases occurring in childhood terminate fatally.

2. The primary diseases of which bronchopneumonia is a complication. These are too numerous to be discussed in detail.

Treatment. The treatment of bronchopneumonia is hygienic and symptomatic. Some authorities are loud in their praises of out-of-door treatment, such as is used in

tuberculosis and in hospital practice in lobar pneumonia. In summer this is unquestionably indicated; but in winter, as many patients with bronchopneumonia are run down and debilitated, care and caution must be exercised. Most physicians prefer a rather warm room, 65° to 70°, if necessary the air being kept moist with steam.

La Fêtra says: "Cold air is particularly indicated in cases with little bronchitis and during convalescence; while during the acute congestive stages of pulmonary infection, with considerable bronchitis, warm and moist air is preferable."

Flannel is usually worn next to the skin. Moderately high fever usually needs no interference; but when signs of toxemia are present, such as restlessness, headache and delirium, the temperature must be artificially lowered. Water in some form is the usual method, as, especially in young children, antipyretics are not considered advisable. Cold sponges, wet packs and cold compresses over the chest (Priesnitz applications) are relied upon. The latter are made by wrapping the chest with one layer of flannel that has been wrung out of water at room temperature, and covering this with three or four layers of dry flannel. This form of hydrotherapy is efficient in lessening nervous symptoms, and also to a lesser extent in lowering fever. Cyanosis must be met by stimulation of the heart if that organ is weak, and by trying to divert the blood to the surface of the body. For this purpose a mustard poultice applied to chest and back is good, but the nurse must be very careful not to leave this on too long, as it may greatly irritate the skin of a young child. The flannel applications to the chest, above referred to, are often of service. Older children should have their position frequently changed, and

babies should be taken up and carried about from time to time. For bad and persistent cyanosis, oxygen inhalations are of value.

The heart often needs stimulation. Alcohol, now so frowned upon generally in medicine, is still conceded a place in the treatment of heart failure in bronchopneumonia. Brandy is the favorite form in which it is given. Other stimulation varies in no way from that given for heart-failure in other acute diseases. Food should be liquid or semi-solid, depending upon the age of the child.

Cough and pain, that call so insistently for treatment in lobar pneumonia, rarely require special measures in bronchopneumonia. If very distressing, codeine is effectual, though in young children the use of opiates in any form is usually avoided as long as possible.

Convalescence must be guarded, as these patients are prone to relapse.

The treatment of bronchopneumonia in the aged presents no distinctive features. Stimulation must frequently be resorted to, and the problem is mainly that of building up the patient's strength and trying to overcome the primary disease of which bronchopneumonia is a complication. Particular attention must be given to changing the position of the patient frequently to prevent hypostatic congestion. In the aged, alcohol seems to have a particularly beneficial effect in keeping up the strength and in enabling the debilitated individual to fight the infection.

CHAPTER XX.

INFLUENZA.

INFLUENZA is an acute infectious disease caused by the influenza bacillus of Pfeiffer (discovered in 1893). The disease is of frequent occurrence, may become epidemic, and in a few instances has become pandemic. The greatest pandemic of recent years was that of 1889-90, and it is interesting to read a quotation from Dr. F. T. Lord showing the enormously rapid spread of the disease:

"The origin of this pandemic (1889-90), like many others, is uncertain. The outbreak in Hongkong in the fall of 1888, in Buckara in the middle of May, 1889, or in Tomsk in the beginning of October, may have been the starting point of the epidemic which occurred in St. Petersburg (now Petrograd) toward the end of October. By November the disease had swept through Germany and France; by December through Austria, Sweden, Denmark, Switzerland, Italy, Spain, Portugal, Belgium and the Netherlands, England, the Balkan States, and North America. By March it had reached India and Australia; by April and May, China and the Gold Coast of West Africa. Berlin was invaded the middle of November; Paris from the 17th to the 20th of November; London the second week of December; Boston and New York the 17th of December. Within a year it had visited nearly all parts of the world."

The great mass of evidence is in favor of the direct transfer of the disease from person to person. In pandemics 40 per cent. of the population may be attacked— a record equaled by no other disease. Infants and the

aged seem less disposed to infection than others. The disease may occur any time, but is most frequent in the fall and winter months.

Symptoms. The incubation period varies from a few hours to a week, the average being from 2 to 3 days. In a typical attack the onset is sudden, with a chill, fever up to 102° or 103°, accompanied by the usual symptoms. Headache is marked and intense. Malaise, pains throughout the body, and weakness and prostration are striking —more so than in almost any other disease, and are out of all proportion to any abnormal conditions discoverable in the patient. The temperature continues for from 3 to 10 days, and drops gradually, weakness persisting well into convalescence, which may be very tedious and protracted.

There are three types of influenza, and it has been noted that in an epidemic a majority of the cases will conform to one of these types.

1. *Respiratory Type* (most common). There is a catarrhal inflammation throughout the entire respiratory tract. There is rhinitis, conjunctivitis, with the accompanying "running" eyes and nose, tonsillitis, with, in severe cases, enlargement of the glands of the neck, laryngitis with hoarseness and tracheitis, accompanied by a burning feeling back of the sternum. Bronchitis sets in, first with a harsh dry painful cough. Soon sputum appears in which the influenza bacillus can be found. Bronchopneumonia may set in, seldom before the 4th day of the disease, and shown by an increase in temperature, pulse and respiration-rate, and by the presence of the characteristic signs in the lungs.

2. *Nervous Type*. This consists simply in an aggravation of symptoms of nervous origin, causing them to be

the most dominant ones.   Among these symptoms should
be mentioned:

Headache. Persistent and intractable.

Peripheral neuritis.

Paralysis of eye muscles.

Meningitis. Very rare.

3. *Gastro-intestinal Type*.   Not well defined.   Here
again the dominant symptoms are these referable to the
abdomen; they include a coated tongue, nausea and vomit-
ing, a tender abdomen, and at times intestinal bleeding.

Mortality.   Influenza uncomplicated or uncompli-
cating is practically never fatal.   Thus, in the German
army, of 55,263 cases only 59 died of influenza, 42 with
pulmonary symptoms.   An influenza pneumonia is always
serious.   The mortality varying from 17 to 50 per cent.
The pre-existing condition of the patient is always of
great importance.   Influenza often proves fatal when
complicating the following diseases:

Pulmonary tuberculosis.

Heart disease.

Nephritis.

Diabetes.

Treatment.   The treatment of the disease is wholly
symptomatic.   Bed and liquid diet during the height of
the fever.   Free catharsis; an ice-cap to the head for
pain.   The use of acetylsalicylic acid (aspirin) and the
coal tars, such as phenacetin, antipyrin, etc., are the
chief measures observed.   For the distressing cough,
some opiate such as codeine or heroin added to an ex-
pectorant mixture is of value.   During long and tedious
convalescence every effort must be made to feed the pa-
tient abundantly, and to zealously protect him from other
infections.

CHAPTER XXI.

TYPHOID FEVER.

TYPHOID fever is essentially a general infection with the typhoid bacillus manifesting itself by a continued fever, a skin eruption, and intestinal ulceration of greater or less severity. The fever runs an average course of from three to four weeks, and terminates by *lysis, i.e.,* gradual descent. Typhoid fever presents greater variations in its manifestations than any other acute infectious disease, and may be complicated by conditions affecting practically every organ in the body. In no disease is careful nursing, conscientious observation, painstaking attention and correct interpretation of changes as necessary and valuable as in typhoid fever. The nurse that has had extended experience in the care of this disease alone, should be well qualified to assume responsibility in the vast majority of medical cases.

HISTORY.

In the writings of Hippocrates, Galen and others of the ancients, it is not possible to distinguish typhoid as an entity. Typhus and typhoid, the plague and, perhaps, malaria and relapsing fever, were apparently grouped together. The greatest difficulty seems to have lain in the separation of typhoid and typhus fevers.

It would be wearisome to give a list of all those quoted in the history of this varied disease, but somewhat detailed mention must be made of Willis, who described an epidemic occurring in 1643 which leaves no room for

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doubt as to its identity with typhoid fever: "Among the features he describes were headache, nosebleed, delirium, an eruption like flea-bites, diarrhea, abdominal distension, intestinal hemorrhage, incontinence of urine and feces, emaciation in prolonged attacks, the long course and the slow recovery without crisis, or the gradual progress to a fatal issue."

"In the history of one patient he describes what was probably an instance of death from perforation: 'Pains and torments cruelly infected his belly, that crying out and moaning night and day, he sent forth his most heavy complaints; his hypochondria and abdomen were tumid like a tympany, and mightily distended.' "

Willis made the observation that the contagion of this disease was slow, but that gradually a household or a community might be infected, and mentions that some of those nursing the patients contracted the disease after a time. He appears to have clearly separated typhoid fever from the plague and typhus fever, and to have appreciated in a remarkable way many of the clinical manifestations as well as the features of epidemics.

Among those following Willis that wrote of diseases that were probably typhoid should be mentioned Sydenham, Baglivi, Hoffman in Halle in 1699, and again in 1728, Huxham of Plymouth, who in 1737 "had taken notice of the very great difference there is between the putrid, malignant (typhus) and the slow, nervous (typhoid) fever." Riedel in Germany wrote in 1748 of typhoid under the name of Darmfieber (intestinal fever); and Röderer and Wagler studied an epidemic in Göttingen (1757 to 1762) which must have been one of typhoid.

More modern descriptions of the disease date from 1813 to 1850, and claim France and our own United

States as the countries of their origin. In 1813 Petit and Serres described "entero-mesenteric" fever. It is worthy of note that in 1824 our countryman, Nathan Smith, published a description of typhoid fever. He did not distinguish between typhoid and tyhus, for he evidently saw only one of the two diseases, but his description stands as one of the classics of American medicine, and is one of the best early accounts of the disease.

In 1829 Louis' great work appeared in which the name "typhoid" was first used. At this period typhoid alone prevailed in Paris, and it was universally believed to be identical with the continued fever of Great Britain, where, in reality, typhoid and typhus coexisted. The intestinal lesion was at that time regarded as an accidental occurrence in the course of an ordinary typhoid. Louis' students, returning to their homes in various countries, studied the disease thoroughly. One of them, Gerhard, of Philadelphia, recognized the prevalence at home of the same disease that he had studied with Louis in Paris, and to him, by the publication of his articles in the "American Journal of the Medical Sciences" in 1835 and 1839, is due the great honor of having first clearly laid down the differences between typhoid and typhus fevers, and of having established a separate individuality for the former.

Almost simultaneously (1833) James Jackson, Jr., of Boston, demonstrated in his father's wards at the Massachusetts General Hospital the identity of the so-called typhus of this country with the typhoid of Louis. In 1838 and 1839 James Jackson, Sr., and Enoch Hale published articles which, together with Gerhard's contributions, served to make typhoid well known in the United States before it was recognized abroad as a clinical and

pathological entity. Shortly thereafter, the studies of Griesinger in Germany, and of Murchison and Tenner in England, did much to spread knowledge of the disease.

Two additional facts are of historical importance. The first of these is, that clear views on the modes of infection were first published by Budd, of Bristol, in the London "Lancet," 1856-1860. He believed that the infective agent was to be found in the stools of the typhoid patient, and that the disease never arose spontaneously, but always from a specific source. He held that a previous focus was necessary before a neighborhood could be infected, and by the study of many epidemics recognized the result of the introduction of infection into a community, and noticed that a few straggling cases might later be followed by a larger outbreak. He considered that an exceedingly small amount of infective material was able to convey the disease, and, arguing from this belief, he put forward the view that the possibility of infection could be prevented if the stools were thoroughly disinfected. His views are essentially correct; and Budd may be regarded as the first to recognize the leading points in the transmission of the disease. The second point to be mentioned is the discovery of the specific germ causing the disease—the typhoid bacillus, or bacillus typhosus, by Eberth, in 1880.

"Typhoid fever occurs in the tropics, and at far northern and southern latitudes, at sea-level and in the mountains, in the city and in the country, and practically everywhere man may go, and local conditions do not prevent the dissemination of the disease. The bacillus typhosus has about the same limits of latitude and longitude as man himself, and no country or race is known to be immune from the disease."

Schuder, in a study of 638 epidemics from 1870 to 1899, found the infection to be carried by water in 71 per cent. In Hamburg from 1885 to 1888 there were 15,804 cases of typhoid fever. The water supply at that time was obtained from the Elbe, not far from where the sewers discharged. The neighboring city of Wandsbeck, with a different water supply, was practically free from the disease. In Paris, with a better water supply, the death-rate from 1882 to 1902 due to typhoid fever was reduced from 142 to 17 per 100,000 population.

These few figures show the paramount rôle played by water in carrying infection. The brief historical summary is interesting as setting forth the early gropings for a clue to this universal scourge, and as showing the effect of the breaking forth of the light of knowledge and the gradual comprehension of the value of a strict prophylaxis in limiting the ravages of a disease that even now exacts a yearly toll of over 25,000 lives in the United States alone, and that is wholly and completely preventable.

Etiology. The essential cause of typhoid fever is the typhoid bacillus, and that alone. This bacillus is motile, flagellated (possessing hair-like processes), grows best in bouillon or milk, is very resistant to cold, being able to live for three months in ice, is usually killed by a temperature of 60° C. (140° F.), is always present in the stools, and usually in the urine.

Modes of infection:

1. *Direct.* By contact with the stools or urine containing typhoid bacilli. The hands becoming contaminated, bacilli are easily taken into the mouth, and so into the intestinal canal. Carelessness with bedclothes, bedpans, urinals, or any articles or utensils coming in con-

tact with the excreta of the patient may very easily cause attendants to acquire the disease.

2. *Water.* This is by far the most common source of all typhoid epidemics. If the water supply of a community is infected, practically every one not immune to the disease is taken ill. Carelessness in disinfection and disposal of excreta, or an inadequately protected watershed, pave the way for an infected water supply. The accompanying diagram illustrates schematically how a water supply may become infected.

Fig. 7.—Diagram to illustrate water supply of town. 1, town; 2, water reservoir; 3, house with typhoid patient—no care taken to disinfect stools; 4, spot where stools and urine are emptied; 5, arrows showing hillside down which bacilli are washed by rain, until infected rain-water reaches reservoir which in turn becomes polluted; 6, pipe running from reservoir to town.

While the diagram shows how the water supply to a whole town may become infected, there are many other ways in which water may indirectly prove to be the channel of infection. Some of these will be briefly mentioned.

3. *Food:*

(a) *Milk.* Often contaminated through infected water used to wash the cans, or through the infected fingers of the milkman, in whose home a case of typhoid fever exists. Fre-

quently it has been found that most cases of typhoid developing in a town are on the route of one particular milk-wagon.

(b) *Shell-fish.* May carry infection either by having lived in contaminated water or by having been shipped in water infected with typhoid bacilli.

(c) *Vegetables.* May carry infection by being washed in contaminated water.

4. *Flies.* These pests are a great source of the communicability of typhoid fever. They alight on the bacillus-laden typhoid stool, and fly away, carrying typhoid bacilli on their feet, to deposit them on any article of food they may choose for a resting place, or any glass of water upon which they may settle. The water or the food, upon being taken internally, infects the individual with typhoid fever.

5. *Clothing.* Especially all articles in contact with typhoid patients, such as night-gowns, towels, sheets, pillow-cases, blankets, handkerchiefs, etc., which, unless carefully disinfected, are fruitful sources for the spread of the disease, as they easily become contaminated through contact with stools or urine.

Pathology. The discussion of this subject will be limited to a consideration of the intestines and spleen, as it is in these two organs that the most characteristic changes are found.

Intestines. These are often distended, and the peritoneum over the bowel may show many small hemorrhagic areas. Ulcers are to be found in the walls of the lower part of the jejunum and ileum, the long axis of the ulcers being parallel with the long axis of the bowel.

A rather detailed consideration of the intestinal ulcera-

tion is necessary, in order that the mechanism of two of the most important complications of typhoid fever (perforation and hemorrhage) may be clearly understood.

There are four stages in the evolution of the typhoid ulcer in the intestinal wall:

(1) Hyperemia and hyperplasia.
(2) Necrosis and sloughing.
(3) Ulceration.
(4) Cicatrization.

1. *Hyperemia and hyperplasia.* This condition is characterized by an intense congestion, and by an increase in the cells (especially the "lymphoid" cells) in the bowel wall, which occurs in the lower part of the jejunum and in the ileum, sometimes even extending into the large intestine. Peyer's patches, which are collections of lymphoid cells normally existing in the bowel wall, become greatly enlarged.

2. *Necrosis and Sloughing.* A death of tissue in the bowel over the affected areas (especially the Peyer's patches or plaques) now occurs. This is due to three factors:

(1) Diminished blood-supply to the involved area due to pressure on the blood-vessels running in the bowel wall.
(2) The formation in these vessels of clots known as thrombi, which plug the vessels completely.
(3) The specific poisoning action of the toxins of the typhoid bacillus.

Necrosis may be superficial or deep.

3. *Ulceration.* When necrosis is complete, the slough begins to separate from its base, and an ulcer results. The separation begins at the edges, and extends inward,

until the entire slough is cast off. (Perforation occurs most frequently at the time of the separation of the slough). Several neighboring ulcers may unite, forming one huge ulcerated area.

4. *Cicatrization.* This is the stage of repair and recovery. It begins as a thin film of granulation tissue covering the base of the ulcer, and gradually extends until all signs of damage have disappeared. After the reparative process is complete, the area is usually deeper than the surrounding tissue, and lighter in color.

The various stages in ulcer formation and repair are to be found in the bowel at one and the same time. Thus, at the height of the disease an ulcer in the stage of repair may be near one

Fig. 8.—Small intestine. Peyer's plaques showing tumefaction and superficial ulceration. Typhoid fever. (From the Pathological Museum of McGill University.)

that has just reached the stage of necrosis and sloughing.

Spleen. This organ is always swollen, usually to 3 times its normal size, due to a tremendous increase in

the lymphoid tissue, of which it is so largely composed.

Symptoms. The incubation period in typhoid fever is very variable—three to twenty-three days. Roughly speaking, about two weeks.

Onset. This is gradual, the patient feeling below par for a week or ten days, suffering from headache, loss of appetite, lassitude, increasing weakness and general malaise. Nosebleed often occurs.

Course. The course of typhoid fever is usually described by weeks:

1st Week. The patient feeling badly, finally takes to his bed. The temperature rises daily in a step-like manner (see chart), beginning with a maximum of 100°, and at the end of the week reaching from 102° to 104°. The face is flushed, the eyes bright. There is considerable headache, and complete loss of appetite. The tongue is coated with a white fur, and frequently clean at the edges. The abdomen is moderately distended, and abdominal gurglings are frequent. There is some mental slowness. The pulse is full and bounding, and is *slow* as compared with the height of the temperature, running usually between 80 and 90 to the minute. Cough with some mucoid expectoration is common. The bowels are usually constipated, though often there is a simulated diarrhea, due to the giving of purgatives. (This must not be confounded with "typhoidal diarrhea," to be mentioned later). At the end of the week rose-spots (the eruption of typhoid fever) appear either on the abdomen, chest or back. They may be few in number or very abundant. They are small, pinkish spots, slightly raised above the surface of the skin, disappearing at once on slight pressure, but reappearing instantly the pressure is released. The spleen is usually enlarged.
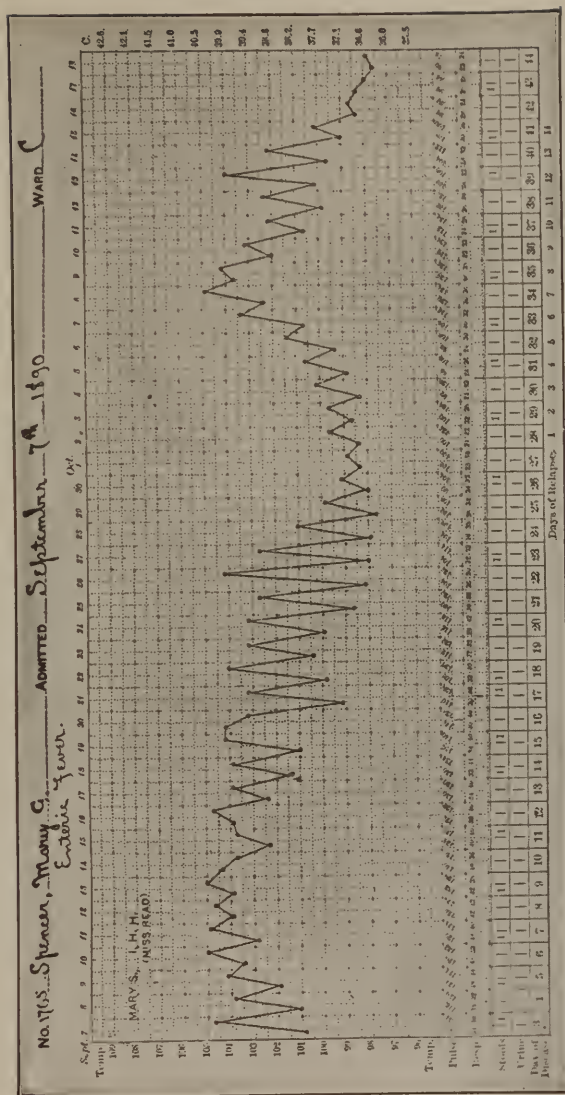
Fig. 9.—Typhoid fever with relapse. (Osler.)

2d Week. During this period of the disease the symptoms mentioned in the preceding paragraph (with the exception of headache, which usually disappears) become more pronounced, and some new ones make their appearance. The temperature on the whole is higher, running between 102° and 105°, and there are fewer remissions. The abdomen may be more distended, and in severe cases, diarrhea may set in. The mental condition is distinctly more dull than in the first week, the patient lying quietly, usually being able to answer questions, but paying very little attention to what goes on. Especially do patients lose the idea of time, and hence will frequently assert that no food has been given for ten or twelve hours, when as a matter of fact such is not the case. The pulse becomes more rapid (90 to 110), and is often dicrotic. Complications may set in.

3d Week. In mild attacks the patient's condition begins to improve (see 4th week). In severe cases the disease is now at its height. The temperature runs steadily high (103° to 105°), the abdomen is greatly distended, the tongue is brown and cracked, sordes appear on the lips, the mental condition is profoundly affected, some patients being wildly delirious, others lying in a stupor. The condition known as "coma vigil" may be present, the patient lying with eyes wide open, staring, and wholly unconscious. There may be picking at the bedclothes. All these symptoms denote intense toxemia, and, taken together, are often referred to as the "typhoid state." Emaciation is extreme. The pulse is rapid (120 or more), weak and thready. There often is diarrhea and incontinence of urine and feces. Complications, especially perforation and hemorrhage, are particularly frequent at this time.

4th Week. If the patient is to survive, improvement usually shows itself. Among the first signs are greater remissions in the temperature (these occurring usually in the morning hours) and betterment in the mental condition, which denotes a lessening of the toxemia. At times the temperature may begin to drop, but the mental condition fails to clear up; this is a bad sign, denoting intense systemic poisoning. The diarrhea lessens, as does the abdominal distension; the pulse becomes slower and stronger; the dirty, foul tongue clears up, and all symptoms abate. By the end of this week convalescence is under way.

In the case of average severity, the patient will be in bed about six weeks; mild cases may be up and about in a little over a month; while severe cases may be confined to bed for three months and more.

The Urine. The urine in typhoid fever presents no characteristic features other than those usually present in any acute febrile disease, save that the diazo-reaction of Ehrlich is usually present. During the course of typhoid a very large amount of urine is frequently passed, due to the large amount of water forced upon the patient.

The Blood. The blood in typhoid fever also presents no notable changes. The most important fact in connection with the blood is that the *leucocytes are not increased in number,* typhoid being one of two common infectious diseases characterized by no increase in those cells. The other disease is tuberculosis. In serious cases there is, of course, a moderate degree of anemia.

A word must be said concerning the *"Widal reaction"* of the blood. This reaction depends upon the presence in the blood-serum of substances known as "agglutinins" (see chapter on Immunity), which have the power to

cause micro-organisms to lose their power of motion (if it exists), and of causing them to agglutinate or "clump." In a patient with typhoid fever specific typhoid agglutinins are present which will cause typhoid bacilli, *and no others,* to clump, and to become motionless.   If, for instance, 40 drops of a broth culture of typhoid bacilli are mixed with 1 drop of the patient's blood-serum, and within an hour the bacilli are seen under the microscope to lose their motility and to clump, the Widal reaction is said to be positive in a dilution of 1 : 40.   If 100 drops of culture are mixed with 1 drop of serum, and the reaction is present, it is said to be positive in a dilution of 1 : 100, etc.

The Widal reaction is an extremely important diagnostic point in typhoid fever.   It is almost never present until the end of the first week, sometimes not until the end of the second week, almost always by the third week ; hence it is of no value at the very beginning of the disease.   If the patient has been inoculated with antityphoid vaccine within three years, the reaction loses its value, as it will almost invariably be positive whether the patient is suffering from typhoid fever or not.

The Stools.   If the stools are not loose, there is nothing about them that is characteristic.   If they are loose, they are generally known as "pea-soup" stools.   They are thin, of a brownish color, and have a peculiar, characteristic odor.   On standing, they separate into an upper fluid layer and a lower, more solid layer.   Constipation is the rule in typhoid, and constipated patients seem to do better.   The early diarrhea is usually due to the giving of purgatives before the diagnosis has been made: the late, or typhoidal diarrhea is to be classed as a complication.

Complications. All the complications of typhoid fever cannot be dealt with here, for to do so would require almost a separate volume. Those to be mentioned include the most frequent and important ones, and those with which the nurse will most often be called upon to deal.

Hemorrhage. Intestinal hemorrhage is caused by the erosion or eating away of the wall of one of the blood-vessels in the bowel by the ulcerative process going on in the intestine. Hemorrhage occurs in about 7 per cent. of all cases of typhoid fever. The amount of blood lost may vary from a few cubic centimeters to a quart, depending upon the size of the injured vessel. When hemorrhage is large it is a serious complication. Hemorrhages may occur singly or there may be many in rapid succession. As a rule, several small hemorrhages prove more serious than one moderately large one, for the amount of blood lost is greater, and there is less time for recuperation. Hemorrhages usually occur at the end of the second week and during the third week of the disease, but may occur at any time from the onset to convalescence.

The nurse, being with the patient continuously, is the one that, far more than the physician, is present at the moment the hemorrhage occurs, and should train her powers of observation in order to suspect the presence of hemorrhage at the earliest possible moment.

Symptoms. A small hemorrhage, a couple of ounces or so, as a rule, gives no symptoms, and is not even suspected until the blood is passed in the stool. With a larger hemorrhage there may or may not be abdominal pain at the time of bleeding. If the hemorrhage is copious there is usually a sudden fall in temperature, a

sense of weakness, and, in marked cases, pallor, with a cold sweat, restlessness and "air-hunger," the patient complaining of a stifling sensation, and restlessly tossing the head from side to side on the pillow. There is usually a change in the pulse at the time of bleeding; it becomes faster, and has a peculiar "bounding" feel, difficult to describe but easy to recognize when once its significance has been appreciated. The main thing for the nurse to note is the *change,* not alone in the pulse, but in the patient's general condition.

The above symptoms permit only the *suspicion* of hemorrhage. *Proof* comes when the blood is passed from the bowel. Usually the blood is dark and clotted, though if the hemorrhage is profuse and low down in the bowel the blood passed may be a brighter red. It is only by constant watchfulness that the nurse will be able to detect the early signs that are suggestive of hemorrhage; but by their prompt detection and interpretation, and by carrying out at once the provisional orders left by the physician, she will do much towards aiding her patient in his fight for life.

Perforation. Perforation is the most serious and most dreaded of all the complications of typhoid fever. It is brought about by the eating away, through deep ulceration, of the peritoneum covering the bowel. Two minutes after the perforation has occurred, the infectious bowel contents are flowing freely into the peritoneal cavity, setting up an acute general spreading septic peritonitis. In the absence of prompt diagnosis and speedy surgical interference, death is inevitable.

No nurse is ever required or desired to make a diagnosis of perforation, but she must be on the alert for those symptoms suggestive of the condition, in order that

she may at once summon the physician. If she is in doubt, she should always call the doctor. It is better to send for him a dozen times on false alarms than to hesitate and delay when the real danger is present, for in cases of perforation minutes count.

Perforation occurs in 2 per cent. to 3 per cent. of all cases, and usually during the third week, though, as in the case of hemorrhage, it must never be forgotten that it may occur at any time.

Symptoms. By far the most important single symptom of perforation is *sudden severe paroxysmal abdominal pain*. Nausea and vomiting, sweating and signs of collapse may occur. Any sudden and obscure change in the patient's condition is suggestive. After perforation the pulse tension *rises* (the opposite from hemorrhage), and eventually the "wiry" pulse of peritonitis is to be felt. From the standpoint of the nurse, *pain* is by far the most important symptom. The leukocytes are increased in number with the development of peritonitis, which is an important aid to the physician in making the diagnosis, as in uncomplicated typhoid there is no leukocytosis. If surgical intervention is not resorted to, the symptoms of general peritonitis will manifest themselves.

Thrombosis. This complication frequently occurs, usually in the femoral or iliac vein. When in the femoral vein, there is pain down the leg, usually some swelling, edema and cyanosis, and the vein may be felt as a hard, tender cord. When thrombosis occurs in the iliac vein, the pain is abdominal, and owing to its severity may suggest perforation.

Lobar Pneumonia. This disease may occur as a complication of typhoid fever. Attention is called to the

chest by pain, cough, and rusty sputum. The temperature may rise, though if it is very high at the time of the onset of pneumonia no change may be observed. The leukocytes are increased in number.

Myocarditis. This condition occurs in a measure in practically every severe case of typhoid fever where the patient is profoundly poisoned and the heart suffers from the effects of toxemia. The symptoms are not characteristic, weakness and some irregularity of the pulse being the main signs to be detected by the nurse.

Tender Toes. Frequently at the height of the disease, or during its latter portion, the tips and under surface of the toes become exquisitely tender, so much so that the weight of the bedclothes produces intolerable suffering. The condition is probably due to an irritation of the sensory nerve-endings.

Abscesses. These occur frequently, and no part of the body is immune from invasion. Typhoid bacilli are sometimes found in the pus. The symptoms vary so widely with the location of the abscess that no detailed description can here be given. In almost every case there is local pain, heat, redness and swelling (if the abscess is on the surface of the body), a rise in temperature, and at times the appearance of chills and sweats.

Diarrhea. This occurs at the end of the second or during the third week, and is a serious complication, being due to profound toxemia and deep intestinal ulceration. The stools may number from four to ten per day, and because of their frequency prove a great additional drain upon a patient already overwhelmed with poison.

Relapse. By relapse in typhoid fever is meant a recurrence of the symptoms of the disease after the temperature has been normal for five or six days. The relapse

resembles in every way the original attack, save that it is usually much shorter and milder, though in no sense free from danger, as it attacks a patient already exhausted. A new crop of rose-spots may appear, the spleen again becomes enlarged, and any of the complications of typhoid, whether present before or not, may occur.

Prognosis. The forecast in typhoid fever must always be guarded, the saying of Hippocrates being very appropriate: "In acute disease it is not safe to prophesy either death or recovery."

Death-rate is highest under 2 years of age.

Death-rate is lowest from 2 to 15 years of age.

Death-rate is lower from 15 to 25 than from 25 to 40.

Death-rate is higher over 40.

Generally speaking, the mortality is from 7 per cent. to 10 per cent.

The following elements are to be considered in estimating the patient's chances for recovery:

1. Toxemia. If it appears early, it is serious. If the patient refuses nourishment, and especially water, the outlook is grave.

2. Nervous symptoms—delirium, etc.—if occurring early, are of bad omen.

3. Pulse. Any rate over 120, save if very temporary, is a bad sign. Irregularity is always serious.

4. Lung complications. Pneumonia is very fatal.

5. Abdominal distension (meteorism), if marked, is a bad sign, as it indicates intense toxemia.

6. Diarrhea. Always a bad sign, indicating severe toxemia.

7. Hemorrhage. Needs no further discussion.

8. Perforation. "Recovery without operation is hardly to be credited,"

Prophylaxis. General municipal measures for the control of the typhoid situation and for the prevention of its spread, when once present, do not concern us here.

SPECIAL MEASURES IN CONNECTION WITH THE PATIENT.

Typhoid fever is a preventable disease, for every typhoid bacillus is within our power and under our control *at the time it leaves the human body*. Typhoid fever does not originate spontaneously, and every case must come from a pre-existing source. Consequently, if every typhoid bacillus were destroyed at the time it is cast off from the human body, the disease would soon be almost eradicated.

The preventive measures, presently to be enumerated and described, concern the nurse more than anyone else for two reasons: first, for her own protection; second, because she is the one that must carry them out, and it is due to her conscientiousness and never-slacking attention that the measures prove efficient. The physician in charge of a case leaves his orders as to prophylactic measures; but if the nurse does not whole-heartedly and scrupulously execute them, they are practically of no value.

(*a*) Isolation. While of course this is not as necessary as in measles or diphtheria, yet the patient with typhoid fever should be alone, save for the nurse, as much as possible. There is no greater mistake than to allow members of the family to come in, sit down and talk with the patient simply because the case is not a desperate one. The room should be arranged with due regard to attractiveness, but all heavy window draperies, carpets, etc.,

should be removed. There should be nothing in the room that cannot be easily and thoroughly cleaned.

(*b*) Disinfection of the following:

    (1) Stools.

    (2) Urine.

    (3) Sputum and vomitus.

    (4) Clothing and bedding.

    (5) Bedpans, urinals, thermometers, syringe-nozzles, etc.

1. Stools. Each physician has his own choice of the particular disinfectant to be used. The following are the agents most in use at present:

(1) Bleaching powder	3	per cent. solution
(2) Milk of lime	1.8	" " "
(3) Cresol	1	" " "
(4) Carbolic acid	5	" " "
(5) Formalin	10	" " "

No matter which is used, the stool should be received into a bedpan containing some of the germicide, and after the patient has finished with the bedpan, enough of the disinfectant should be added to secure twice as much disinfectant as there is stool. Solid clumps of feces should be broken up with a rod, and the whole mass thoroughly stirred and set aside, protected from flies, for two hours before being disposed of.

2. Urine. Bichloride of mercury is the best disinfectant. A 1 : 1000 solution is used, there being at least $\frac{1}{40}$ as much of the bichloride solution as there is urine; thus, 1 ounce of bichloride solution will disinfect 40 ounces of urine. It is best to keep the bichloride solution in a large jar, and pour the urine into that from the bed urinal, the jar being emptied daily. The mixture of bichloride and urine should stand for at least two hours before being thrown away.

3. Sputum and vomitus. Neither of these is ordinarily obtained in typhoid fever, but when present the sputum should be received in a sputum cup and burned, while the vomitus can be disinfected with the same germicide used for the stools.

4. Clothing and bedding. Gowns, sheets, pillow-cases, etc., can be soaked in 5 per cent. carbolic acid or 10 per cent. formalin for two hours; then boiled. Rubber sheets to be soaked in carbolic, as boiling is injurious to them.

5. Bedpans, urinals, etc. Fill pans or jars with agent used for stools, then scald in water. Boil all enema tubes, syringes, nozzles, etc.; keep the thermometer in a glass or small bottle containing carbolic 5 per cent. or formalin 10 per cent.

See to it that the patient has separate dishes, glasses, silverware, napkins. If possible, select some dishes and silver of a different pattern from that used by the rest of the household, in order to prevent mistakes occurring.

PRECAUTIONS ON THE PART OF ATTENDANTS.

Great care is an absolute necessity. A basin of bichloride (1 : 1000) should be at hand, as well as plenty of hot water and soap. Whenever the nurse has been busy with the patient—bathing, giving an enema, making the bed, cleansing the buttocks after a stool, etc.—she should carefully wash her hands with water, soap and a brush, and then immerse the hands for three minutes in bichloride. Remember that every germicide takes time to act, and do not be misled as to the efficacy of the pale-blue solution to the extent of believing that dabbing the tips of the fingers therein ensures absolute sterilization. Be sure to have a basin of bichloride and a clean towel for the doctor whenever he calls.

When nursing a case of typhoid fever in a private house. try in every way to avoid having anything whatsoever to do with the preparation or handling of the food for the rest of the family; try, if possible, not to have to go to the ice-chest; have a little refrigerator for the exclusive use of the patient. If such arrangements cannot be made, cleanse the hands with scrupulous care before touching any food whatsoever.

These precautions entail much hard work, but their never-failing observance places the trained nurse where she rightly belongs—in the position of a power for good in the community and in the family; and, in addition, her preventive labors will greatly lessen her own chances of developing typhoid fever.

Preventive Typhoid Inoculation. An active immunity to typhoid fever can be brought about by the injection of dead typhoid bacilli. "The procedure is harmless, rational, and effective" (Rosenau).

Preventive inoculation against typhoid is a procedure to which every nurse should submit.

The vaccine is made from a twenty-four-hour broth culture of typhoid bacilli killed by being heated to 60° C. for one hour. Injections are given every ten days for three doses, between 50,000,000 and 500,000,000 dead bacilli being injected at each dose. There may be moderate evidences of reaction: soreness with pain, heat, redness and swelling at the site of injection, a moderate rise in temperature, or a feeling of general malaise. These symptoms appear within twenty-four hours after the injection, and usually subside within 24 to 36 hours after their onset. The immunity conferred lasts from three to four years, and may be indefinitely continued by further inoculations.

"As a striking instance of the protection offered by vaccination against typhoid may be quoted the result in the United States Army during the maneuvers around San Antonio, Texas, in the summer of 1911.

"All the men, numbering 12,801, were inoculated. From March 10th to May 10th, two cases of typhoid fever occurred, both patients recovering. One patient was a private in the hospital corps who had not completed the series of inoculations, having had but two doses; the other was a teamster who had not been inoculated. Among the 12,801 men there were but 11 deaths from all diseases. Typhoid fever was prevalent at this time in the neighborhood. In the city of San Antonio there were 49 cases, with 19 deaths" (Rosenau).

Treatment. Typhoid fever is a self-limited disease, and we have no means at our command with which to shorten the attack. As in the case of the great majority of maladies, we are unable to treat the disease itself. We must devote our time and care to treating the patient that is suffering from the disease.

While every physician prefers a certain routine treatment in typhoid fever, especially as regards the diet, there are certain fundamental principles so generally accepted and practised that many or all of them will be made use of in by far the greater number of cases.

Rest. Absolute rest in bed in the recumbent position, with use of bedpan and bed-urinal, are essentials throughout the course of the disease, and well into convalescence.

Diet. There are almost as many diets for typhoid as there are physicians treating the disease. The "diets" vary from that of certain German authorities who withhold practically everything save water, to the advocates of the "high-calorie" diet, which is decidedly liberal. The diet in typhoid fever will be dealt with generally, no hard-

and-fast rules being laid down (for none really exist), the author contenting himself with registering his decided personal preference for the more liberal methods of feeding.

Liquid Diet. This is probably the most used. Reliance is placed on the following articles:

> Milk. Four to 6 ounces every four hours, to which are added 2 ounces of lime-water.
>
> Egg-albumen. The whites of 2 eggs every four hours, alternating with the milk.

Thus the patient receives nourishment every two hours.

Many patients will successfully weather an attack of typhoid on these two articles of food. If the milk disagrees, or if the patient tires of it, buttermilk, whey, koumiss, or even peptonizd milk can be tried. Milk may also be taken with ease by many if the taste is disguised by the addition of a very small amount of tea or coffee.

Ice-cream is a very satisfactory food, being nutritious, palatable, and readily taken, especially by children.

Clear soups are permissible, but must be taken in addition to, and not instead of, other food. Their taste is pleasant, but their nutritive value is slight.

Tea and coffee are usually allowed in moderation, unless in the opinion of the physician there exists some contraindication.

Beef broths and artificially prepared foods are, as a rule, not necessary; if the patient can take food at all, he can take natural foods.

While it is an every-day clinical fact that hundreds of patients do well on the scheme of diet sketched above, the advocates of more liberal feeding claim that by their method the patient is less emaciated, less exhausted, and more rapidly convalescent.

Referring to the chapter on "Foods and Nutrition" it will be seen that an average individual weighing 70 Kg. (154 lbs.) needs practically 2200 calories of food in twenty-four hours while in a state of health. When a victim of fever, from whatever cause, the tissues of the body are consumed (oxidized) more rapidly than normally, and consequently an overplus of food is necessary.

Under a strictly milk diet, assuming that the patient takes 2 quarts daily, the twenty-four-hour total is but 1300 calories. The advocates of the high-calorie diet which has been championed and elaborated mainly by Drs. Warren Coleman, of New York, and Shattuck, of Boston, believe in pushing the caloric value of the diet up to 3000 calories in twenty-four hours, and, if this is well borne, even exceeding that amount, sometimes reaching 4000 to 5000 calories during convalescence.

The following table shows the variety of foods allowed, together with their caloric value:

Name	Amount	Calories
Apple sauce	oz. 1	30
Bread	Average slice	80
Butter	1 pat	80
Cereal	oz. 1½	50
Crackers	oz. 1	114
Cream, 20 per cent.	oz. 1	60
Eggs, whole	oz. 2	80
Egg, white	oz. 1	30
Egg, yolk	oz. 1	50
Lactose	oz. ½	36
Milk, whole	pint	325
Potato, whole	medium	90
Potato, mashed	oz. ½	70
Rice, boiled	oz. ½	60
Sugar-cane	1 lump	16
Toast	Average slice	80

With this table at their command, physician and nurse can work together and keep a very accurate record of the actual fuel value of the food the patient is getting. No set rules can be given for the administration of the diet. The patient is to take all he can, but is not to be forced beyond the limits of comfort.

Ardent advocates of this form of diet claim:

No marked emaciation.

No typhoid state.

Many patients able to read and divert themselves during their illness.

Patients able to be up and about sooner, and feel stronger.

A quotation from an article by Dr. Coleman is here appropriate:

"The physician should possess at least a rudimentary knowledge of the caloric value of food. But probably *the chief requisite to the successful administration of the diet is intelligent co-operation on the part of the nurse.** Where a nurse is trained in the use of the diet, general directions regarding the total number of calories will suffice. At her discretion, she will increase or lessen the total amount of food or of particular articles while awaiting further instructions. When a nurse is not trained in the use of the diet, the physician himself must assume immediate control of the feeding."

Water. Equal to, if not surpassing the diet in importance, is the amount of water taken by the patient. Too much water can hardly be given, for by its diuretic action it flushes out the kidneys, and, in addition, by its presence in the tissues it serves to dilute and thus render less

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* Italics are mine.

harmful the toxins of the typhoid bacillus.   Practically all authorities agree that at least 2 quarts of water should be taken in twenty-four hours, and many prefer their patients to take, as nearly as possible, 100 ounces (a little over 3 quarts) daily.   The nurse must exercise vigilance and patience in order to persuade the patient to take the requisite amount of water, but such efforts are well repaid, for water is unquestionably the best "medicine" for typhoid fever.

Hydrotherapy.   While the previous paragraph may well be termed "internal hydrotherapy," "external hydrotherapy" is probably the one most important method of treating typhoid fever.

Hydrotherapy is practised in three ways:

(1) Sponges.

(2) Packs.

(3) Tub baths.

In hospital practice tub baths are generally preferred. In private practice, because of the number of attendants required to give the tub bath, and because of the difficulty in securing a portable tub, sponges and packs are usually resorted to.   The effect of all three is the same, the tub bath being probably the most efficient.

Each physician has his own rule for the indications for hydrotherapy.   In some hospitals the routine order is a tub bath every three hours when the temperature is over 102.5°.   Baths, packs or sponges are given for from ten to twenty minutes, the first ones given being usually shorter.   When the sponge or pack is used, the temperature of the water is usually about 70° F., though that may be altered in each individual case.   For the first tub bath, the temperature of the water is generally not under 85° F., and the bath is never given at a lower temperature than 65° F.

It is not the intention of the author to go into the details of the technique of giving sponges, packs or tub baths, as that more properly comes under the head of "practical nursing," and the teaching of each training school varies in some of the details of the procedure.

Advantages of hydrotherapy:

(*a*) Toxemia lessened—probably the most important feature. Patients practically comatose when the bath is begun can, at the end of ten or fifteen minutes, answer questions fairly intelligently. The "typhoid state" is more rarely seen under the use of the baths. Delirium and tremor are lessened, and there is lessened absorption and increased elimination of toxins.

(*b*) Temperature reduced. Contrary to the general supposition among the laity, reduction in temperature, while desirable and welcome, is essentially *not* one of the chief objects of the baths. At the height of the disease the temperature may be but very slightly influenced (less than one degree), yet the general condition may be very markedly benefited.

(*c*) Circulation. The vasomotor system is stimulated —the general tone of the vessels is raised. The heart-rate is lessened, the pulse is made smaller and harder, and blood-pressure is raised.

(*d*) Respiration. With each bath the patient takes a few full, deep breaths, and thoroughly expands the lungs. This lessens the danger of passive congestion at their bases in the deep hollows present on either side of the spine.

(*e*) Digestion. Disturbances of this function are less common, and the mouth is usually in better condition, due to lessened toxemia.

(*f*) Skin.   Liability to bed-sores is decreased.

(*g*) Mortality is lowered 5 to 7 per cent.

Contra-indications to hydrotherapy: Baths should not be given in the presence of:

(1) Abdominal pain.

(2) Hemorrhage.

(3) Perforation.

(4) Phlebitis.

(5) Great prostration with failing circulation.

(6) Any serious complication.

General Measures. *The care of the mouth* is all-important, and scrupulous attention on the part of the nurse to this disagreeable duty will often result in avoidance of the dry, brown, cracked, fissured tongue, the sordes on the lips, and in lessening the bad taste and general "cottony" feeling of the patient's mouth.

The mouth should be cleansed after each feeding, and special attention should be devoted to the tongue. Some good mouth-wash is desirable, and tooth-picks with the end wrapped in cotton can be dipped in this and rubbed over the teeth and gums. The lips should be frequently moistened with glycerine, vaseline, or some softening and soothing ointment. The care the nurse takes of her patient's mouth is a pretty good index of the general attention that patient is getting.

The care of the skin is also very important. Frequent alcohol rubs should be given, after which a dusting powder should be applied. After each stool the buttocks should be sponged with carbolic acid (1 : 40) solution, and then freely powdered, care being taken to get the powder well into the natal cleft. At the slightest appearance of redness or irritation on the skin, pressure should at once be relieved by means of a rubber or cot-

ton ring, and the physician's attention directed to the irritated area, that he may deal with it as he sees fit.

Frequent change of position is very essential, as it lessens the chances of passive congestion in the lungs, and also lessens the occurrence of bed-sores. Patients that are not very ill will change position of their own free will, but the stuporous patient will lie for days flat on his back. Such individuals must be rolled on the side (first on one side, then on the other) two or three times daily for half an hour at a stretch, and retained in that position by means of pillows, bolsters or sand-bags. See that the patient voids plenty of urine, and that the bladder does not become overdistended, which can easily happen in stuporous patients.

Routine Drug Treatment. Generally speaking, there is none in typhoid, drugs being used only to meet special conditions as they arise. The one exception to this rule is the use of hexamethylenamin (urotropin) in 5-grain doses three times daily to render the urine sterile.

Treatment of complications.

1. *Hemorrhage.* The usual routine is:

Discontinue all food until told to resume it by the physician in charge.

Discontinue stimulants if they are being given.

Give morphia $\frac{1}{4}$ grain with atropin $\frac{1}{150}$ grain hypodermically.

Do not move bowels for three days; then give an oil enema, to be followed by a soapsuds enema.

Other measures as indicated by the physician.

2. *Perforation.* Immediate operation is the only treatment.

3. *Thrombosis.* Place the leg at absolute rest on a pillow. Move only when necessary, and then with the greatest care.

Do not rub the leg, as by doing so bits of the clot in the vein may be detached, float about in the circulation, and by their final lodgment cause the death of the patient.

4. *Failing Heart.* The methods of combating heart weakness are so varied that it is impossible to go into them in detail. Strychnia will be used, as also caffeine, various preparations of digitalis, and in many cases alcohol in the form of whisky or brandy. In the event of sudden collapse, treatment is similar to that given in detail in the chapter on Lobar Pneumonia (*q.v.*).

5. *Meteorism.* A simple diet and plenty of water lessen the occurrence of this distressing condition. When it is present, food is discontinued (save water), and turpentine is administered in the form of stupes, and by enema.

6. *Diarrhea.* Diet cut to albumin water. Drugs as seen fit by the physician. Bismuth and lead acetate those most in use. Starch and laudanum enema sometimes given.

Management of other conditions.

(*a*) *Toxemia.* Water, inside and out, is the best treatment. In addition to the water taken by mouth, salt solution may be introduced by rectum (Murphy drip), under the skin (hypodermoclysis), or into a vein (infusion). It may be necessary to feed the patient by means of a stomach-tube.

(*b*) *Headache.* This condition is prominent usually only during the first week, and is generally best controlled by the use of the ice-bag. It may become necessary to use drugs, such as codeine.

(*c*) *Delirium.* Being one of the manifestations of toxemia, the free use of water is the best mode of treatment. When delirium is continued, active, and is exhausting the patient, a good dose of morphia hypoder-

mically produces the best results. At times delirium is so violent as to necessitate hyoscin, while at others, bromides will control it satisfactorily.

(*d*) *Constipation.* Distinctly desirable. Aside from moving the bowels daily by enema, nothing should be done to interfere with it.

(*e*) *Abdominal Pain.* Must always be looked upon as possibly a symptom of a serious complication. Heat or cold will often relieve. It is not looked upon as wise to give morphia for the relief of abdominal pain in typhoid fever.

(*f*) *Tender Toes.* Remove pressure by making cradle (use barrel hoops if necessary) over the feet so that they will not come in contact with the bedclothes.

Convalescence. When the temperature has been normal for about a week, the patient is usually allowed to be propped up in bed, and three or four days later can be placed in a chair, beginning to walk when strength permits. The appetite of convalescing typhoid patients is proverbially large, and care must be exercised lest they over-eat in their enthusiasm. The individual steps in convalescence vary so with the particular case that their enumeration or description is impossible.

General points in treatment.

1. Absolute rest—as much isolation as possible.
2. Simple, but not necessarily meager, diet.
3. Water in abundance:
    (*a*) Inside.
    (*b*) Outside—packs, sponges, baths.
4. Bowels to be let alone if not too loose.
5. Drugs only for special conditions—none as a routine, save hexamethylenamin (urotropin) to destroy typhoid bacilli in urine.
6. Constant, never-ceasing vigilance.

CHAPTER XXII.

TUBERCULOSIS.

TUBERCULOSIS is an infectious disease caused by the tubercle bacillus. It is the most widespread and the most frequent serious disease of the human race, one death in seven, from all causes, being due to it. Tuberculosis may attack any and every portion of the body, certain organs and structures such as the lungs, lymphatic glands, and joints being particularly susceptible. No matter what organ or structure is attacked, the fundamental cause is one and the same (the bacillus of tuberculosis), and the pathological process is also the same.

Tuberculosis of the glands, joints, bones, etc., are known as cases of "surgical tuberculosis." These conditions will not be considered. In this chapter will be taken up what might be termed "medical tuberculosis," *i.e.,* tuberculosis of the lungs and the most frequent tuberculous complications of a medical nature that are met with.

Historical Note. Pulmonary tuberculosis has been known to man from the most remote times. Babylonian records, the most ancient known, make mention of it. Hippocrates (B. C. 460-376) gives an intelligent description of the disease. Aristotle, a contemporary of Hippocrates, notes that it was a general belief among the Greeks that phthisis or consumption was contagious. No advance was made with regard to the nature of the disease for 1400 years, when anatomical study began. Sylvius (1695) first indicated the connection between

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tuberculosis nodules and phthisis. Morton (1689) brought the tubercle prominently to attention as the true cause of phthisis. Stark (1785) accurately described miliary tubercles, and paved the way for the correct understanding of their nature and relation to phthisis. Bayle (1803) studied miliary tubercles in all stages, stressed the importance of differentiating young from old tubercles by differences in their opacity, and claimed that true tuberculosis is a constitutional affection which can cause development of nodules in all organs, and not originate in inflammation, although often complicated with it.

"Laennec (1819), whose work soon followed Bayle's, consummated and simplified the knowledge thus far gained. He recognized the unity of all phthisis as tuberculosis, and scrofula as tuberculosis of lymph-glands; his ideas in general as to causation and infection were distinctly modern, and his description of the tubercle and its transformation toward ulceration are unexcelled. Most valuable of all was his gift of the art of auscultation. No genius like that of Laennec so far anticipated his own day" (Baldwin).

In December, 1865, Villemin presented his paper "On the Cause and Nature of Tuberculosis and the Inoculation of the Same from Man to Rabbit." His conclusions were as follows:

"1. Tuberculosis is a specific affection.

"2. It has its origin in an inoculable agent.

"3. The inoculation from man to rabbits is very successful.

"4. Tuberculosis pertains, therefore, to the virulent diseases, and should be classed with variola, scarlatina, syphilis, or, better still, with glanders."

Villemin employed many different elements for his inoculation experiments, among them being fragments of lung tubercle, sputum, blood, tuberculous glands, tubercle from cattle (bovine tubercle), and obtained positive results (*i.e.,* development of tuberculosis in the rabbit) in almost all cases.

Finally, in 1882 Robert Koch, Health Officer in an obscure German town, discovered the tubercle bacillus, and proved conclusively that it was the sole cause of any and every form of tuberculosis.

Etiology. The sole cause of tuberculosis is the tubercle bacillus. It belongs to the vegetable kingdom, and when seen in stained preparations appears as a small, straight, or slightly curved red rod.

The tubercle bacillus requires the presence of oxygen in order to develop. It grows best at body temperature (98.6° F., or 37° C.). Temperatures below 30° C. or above 42° C. markedly lessen its growth. Direct sunlight kills the germ in a few hours. Five per cent. carbolic acid kills it in a few minutes; but when the bacillus is embedded in sputum, five or six hours are often necessary to kill all the organisms.

Modes of Infection. It is now the opinion of most authorities that infection with the tubercle bacillus takes place in childhood, usually before the tenth year. The bacilli may gain entrance into the body by two routes:

1. Inhalation—being breathed in with the air.

2. Ingestion—being taken into the intestinal tract with food.

No matter how the bacillus finds an entrance, it quickly goes to lodge in the mesenteric lymph-glands that lie at the back of the abdomen near the spine, or in the mediastinal or bronchial lymph-glands situated in the

chest around and between the roots of the lungs. In these glands the tubercle bacilli may lie for years, and in fact for a lifetime, without causing any symptoms. If, however, for any reason the resisting powers of the body are markedly lowered, the bacilli take advantage of this, and by the action of their poisons, as well as of their bodies, gain the upper hand. They most frequently migrate to the lungs, which are the organs in the body most susceptible to their inroads. Symptoms then make their appearance, and the individual becomes ill with tuberculosis.

It is important to stress the difference between "tuberculous infection" and "tuberculous disease." Every individual harboring tubercle bacilli in his body is the victim of "tuberculous infection." It has been proven from countless autopsies in large general hospitals that, of individuals dying from all causes, over 85 per cent. showed signs of "tuberculous infection." On the other hand, an individual is not the victim of "tuberculous disease" until symptoms appear that warrant a diagnosis of tuberculosis.

Contributory causative factors.

1. Heredity. In the past the influence of heredity was greatly overestimated. We now know that a child born tuberculous (the only way in which the disease can be really inherited) is so rare as to be a curiosity. Heredity, however, is not to be set aside, for it does pass on to the child a *predisposition to infection* with the tubercle bacillus, and also a *lack of resisting power* to the bacillus when once infected. Consequently, individuals in whose family history there is a marked tuberculous strain are far more likely to succumb to the disease than are those whose family tree is unscathed.

2. Environment.   Far more important in determining the outbreak of pulmonary tuberculosis are the environment and habits of the individual.   These can best be considered under several sub-headings:

> (*a*) Dissipation. "Wine, woman, and song" furnish a good soil for the development of tuberculosis.
>
> (*b*) Lack of air in the home, the office or the workshop. Tuberculosis is essentially a "house disease," and prolonged residence in badly ventilated quarters greatly lowers resistance.
>
> (*c*) Overwork. ⎫
> (*d*) Insufficient food. ⎬ All lower bodily resistance.
> (*e*) Smoke and dust. ⎭
>
> (*f*) Prolonged contact with tuberculous individuals that expectorate carelessly and promiscuously.

(*g*) Lack of light and sunshine.   Tubercle bacilli that have been expectorated grow and multiply best under these conditions.

Pathology.

The Tubercle.   No matter where the tubercle bacillus shows its activities, the result is the same—the tubercle.

To the naked eye the tubercle is a small pearly-gray mass about the size of a pin-head.   Tubercles in the affected area may be scattered, and at a distance from each other, or so close to one another as to leave no appreciable space between. Several tubercles may join together or coalesce.   While the young tubercle is pearly-gray, the older tubercles lose this color, and become opaque and whitish in the centre.   This occurs because the toxins of the tubercle bacillus act so as to block off the minute blood-vessels going to the portion of the

organ that is invaded, and thus the blood supply is cut off. In the absence of blood supply there must, of course, be death of tissue. The tuberculous tissue undergoes a process known as "caseous degeneration" or "caseation"—*i.e.,* a degeneration into a cheesy mass, having no definite structure. When this occurs in the lungs to any great extent, the cheesy, decayed matter is thrown off in the sputum, and a cavity is the result, the cavity being nature's attempt at safeguarding the body by getting rid of decayed tissue that is no longer of any use, and leaving in its place a cavity or "hole" which nature again attempts to safeguard by weaving about it a capsule of dense, fibrous tissue, in order to wall it off.

Types of tuberculosis.

Acute General Miliary Tuberculosis. This condition, as the name implies, consists in an invasion of the entire body by the tubercle bacillus. It usually occurs when, from some known or unsuspected focus of tuberculosis, a large number of bacilli are set free at once in the blood-stream. The body has no time to marshall its defensive resources, and is overcome by the extent and intensity of the infection. Miliary tubercles are to be found scattered throughout the body—in the lungs, brain, liver, spleen and other organs.

Symptoms. These are vague, as far as the possibility of diagnosis is concerned, for there are practically none that point to tuberculosis. The picture is one of intense general sepsis, and at first is frequently mistaken for typhoid fever. The temperature is irregular, showing marked variations (in one case seen by the writer the minimum in twenty-four hours was 96° and the maximum 106°); there are frequent chills and abundant sweats, accompanied by rapid and profound emaciation.

The heart action is very rapid (120 or more), respiration is rapid (usually above 30 to the minute), and usually some cyanosis is present. There is *no leukocytosis,* this fact being an important element in diagnosis.

The course of the disease is rapidly progressive, and the outcome invariably fatal. There is no treatment that is of any avail.

Pulmonary tuberculosis.

Clinical Varieties of Pulmonary Tuberculosis. These are three in number:

(1) Acute miliary tuberculosis of the lungs.

(2) Acute tuberculosis. (Galloping Consumption).

(3) Chronic tuberculosis.

1. *Acute Miliary Tuberculosis of the Lungs.* This form of tuberculosis occurs either

(1) As a primary affection.

(2) As a result of dissemination from a pre-existing recognized or unsuspected focus of disease in the lung.

In either case, a large number of bacilli are suddenly set free in the lesser (pulmonary) circulation, so that the pulmonary tissue is bathed in blood rich in virulent tubercle bacilli. The result is the formation at approximately one and the same time of an infinite number of miliary tubercles throughout the lungs. The tubercles are all young, and have the characteristic pearly-gray appearance. Upon touching a lung filled with miliary tubercles, the sensation is exactly as though the lung were filled with bird-shot.

Symptoms. These resemble very greatly those of acute general miliary tuberculosis, and therefore will not

be repeated. Stress must be laid, however, upon two symptoms:

(1) Dyspnea,

(2) Slight cyanosis,

which are practically the only ones that point to the lungs. Most of these patients have no cough, and few of them show expectoration. This form of tuberculosis of the lungs is uniformly fatal. A few cases are cited where the disease has changed into a more chronic type, and where life has been somewhat prolonged, but as a rule death ensues in from six weeks to three months.

The treatment of this type of the disease differs in no way from the management of the ordinary bed case of chronic ulcerative phthisis, and consequently will not be dealt with separately.

2. *Acute Tuberculosis.* In this type of pulmonary tuberculosis an entire lobe of the lung is involved. The picture at first is almost exactly similar to that of acute lobar pneumonia (*q.v.*), and in fact can with difficulty be distinguished from that disease, unless the patient is known to be tuberculous. There is the same high temperature, cough, sputum, pain in the side from pleurisy, dyspnea, and, in very severe cases, cyanosis. Two points worthy of notice are that in acute pneumonia phthisis the sputum is rarely rusty, and that there is no leukocytosis. The affected lobe of the lung is solid (consolidated), as in lobar pneumonia. As time goes on, however, the picture changes. *The expected crisis does not occur;* instead, the temperature remains high, the patient becomes more and more toxic, the sputum becomes yellow, green, and mucopurulent, and, if the tubercles in the lungs have had time to caseate and break down, tubercle bacilli may be found in the sputum.

The course of the disease is either short or protracted. The writer has seen death occur within fourteen days from the onset of the disease, but in many cases the acute stage is weathered, and the disease goes over into the type of sub-acute or chronic ulcerative phthisis with extensive cavity formation. In some cases recovery ensues; others that have been able to live through the most acute stage go on to a life of total or semi-invalidism for months or years.

As in the case of acute miliary tuberculosis of the lungs, the management of these cases differs in no wise from that of bed cases of subacute or chronic ulcerative phthisis, and will therefore not be dealt with here.

3. *Chronic Tuberculosis.* This form of tuberculosis is by far the most common. It is divided into three classes:

(*a*) Incipient.

(*b*) Moderately advanced.

(*c*) Far advanced.

An entirely satisfactory classification of pulmonary tuberculosis has not yet been reached. Attempts have been made to classify the disease according to the amount of involvement found in the lungs, and according to the symptoms presented by the patient. The best classification known to the writer is a combination of these two, which was adopted by the American Sanatoria Association in 1916. Though, strictly speaking, a classification of pulmonary tuberculosis has nothing to do with the duties of the trained nurse, this classification is given, as it can, if carefully studied, give an insight into the many ways in which this disease may present itself.

LESIONS.

Incipient. Slight infiltration limited to the apex of one or both lungs, or a small part of one lobe. No tuberculous complications.

Moderately Advanced. Marked infiltration, more extensive than under incipient, with little or no evidence of cavity formation. No serious tuberculous complications.

Far Advanced. Extensive localized infiltration or consolidation in one or more lobes. Or disseminated areas of cavity formation. Or serious tuberculous complications.

SYMPTOMS.

A. (Slight or none). Slight or no constitutional symptoms, including, particularly, gastric or intestinal disturbance, or rapid loss of weight; slight elevation of temperature or acceleration of pulse at any time during the twenty-four hours. Expectoration usually small in amount or absent. Tubercle bacilli may be present or absent.

B. (Moderate). No marked impairment of function, either local or constitutional.

C. (Severe). Marked impairment of function, local and constitutional.

This scheme is flexible in that it offers the following combinations :

Incipient *A*.	Moderately Advanced *A*.	Far Advanced *A*.
Incipient *B*.	Moderately Advanced *B*.	Far Advanced *B*.
Incipient *C*.	Moderately Advanced *C*.	Far Advanced *C*.

Thus combinations of the local conditions in the lungs and of the general symptoms can be obtained which go far toward placing each individual case in its proper grouping.

Symptoms.

(*a*) Incipient. While the trained nurse will but very rarely be called upon to care for a case of really incipient tuberculosis, she should nevertheless be familiar with the symptoms of this condition, as it is only by spreading their characteristics and their importance throughout every community that the disease can ever be stamped out.

The symptoms of incipient pulmonary tuberculosis are vague and elusive. No one symptom is conclusive; all are but very rarely present; but the combination of any three or four are extremely suggestive.

X. Not pointing to the lungs:

(1) Fatigue. A loss of vigor and of ambition, a tired feeling out of proportion to the amount of work bringing it about, and from which the patient does not promptly recover.

(2) Rapid heart action. Over 85 to 90, especially early in the morning, before arising.

(3) Gradual and persistent loss of weight and strength.

(4) Marked and unaccountable nervousness and irritability.

(5) Loss of appetite and symptoms of indigestion.

(6) Slight afternoon fever—99° to 99.5°—increased by moderate exertion, such as an hour's walk.

Y. Pointing to the lungs:

(7) Cough. Slight, dry, hacking, most noticeable in the early morning.

(8) Sputum. Grayish-white or light-yellow. Slight in amount, usually not exceeding 2 teaspoon-fuls in twenty-four hours.

(9) Dyspnea. Usually slight, transient, and only noticed after some mild physical exertion.

(10) Hemoptysis.

(b) *Moderately Advanced*. The difference in the symptoms of an incipient and of a moderately advanced case is usually one of degree only. The strength of the patient becomes so poor that work is abandoned. The pulse-rate may or may not show a change. The weight

is decidedly below par, and the emaciation of the patient becomes apparent. The temperature is more marked, rising usually from 100° to 101° in the afternoon, and often registering 98.6° to 99° in the morning. The cough becomes increasingly troublesome, and often disturbs the patient's rest. It frequently assumes a looser and more hollow character, and may come on in such paroxysms as to cause vomiting. The sputum becomes more profuse, frequently reaching a total of 2 ounces in twenty-four hours, and, though there are many exceptions to this rule, generally becomes yellow, mucopurulent, and tenacious. Tubercle bacilli are usually present in the sputum. Dyspnea is more marked on exertion, and may even persist when the patient is at rest. Hemoptysis is more frequent than in the incipient stage, and, when it occurs, is apt to be more profuse. *Night-sweats* may put in their appearance, and tuberculous complications, especially tuberculosis of the larynx, are frequent. Disorders of digestion are more frequent and less tractable than in the incipient form.

(*c*) *Far Advanced.* Here, again, the difference in symptoms between moderately advanced and far advanced cases is one of degree only. The weakness and emaciation become extreme, and the patient is often absolutely bedridden. The pulse is usually over 100, and weak. The appetite is bad, and digestion is poor, these patients being greatly distressed by the copious formation of gas in the intestines. The temperature may range from 99° to 103° or more, or else may be typically "septic" in type, rising and falling with no reference to the time of day. Chills, especially in the morning, are frequent, and the nights are rendered hideous by the drenching sweats. Cough is severe, at times almost constant,

deep and hollow, and usually causes the patient to awaken a half-dozen times during the night. The sputum is copious, from 3 to 8 ounces in the twenty-four hours, yellow, or yellow-green, tenacious, often with a sweetish, sickening taste, and at times foul-smelling. Dyspnea is marked, constant, and is greatly increased by the slightest physical exertion. Hemoptysis is frequent, and when it occurs in any large amount is very serious and sometimes fatal. Tuberculous laryngitis, tuberculous enteritis, and tuberculous meningitis are of frequent occurrence, especially the first two. Often, patients can lie in but one position, usually on the relatively sound side, any change bringing on an exhausting fit of coughing. With the wretchedness incident to the high temperature, the cough, the sweats, the inability to eat, the frequent and painful complications, these unfortunates form one of the saddest sights in medicine, and many of them, fully aware of the helplessness of their condition, welcome death as a blessed deliverance.

Modes of death in pulmonary tuberculosis.

1. Exhaustion. By far the most frequent.

2. Hemorrhage. Usually only in advanced cases with large cavity formation.

3. Suffocation. Lung involvement so great that the remaining breathing-space is insufficient to maintain life.

4. Pulmonary edema. Sudden, and often the result of unexpected heart failure.

5. Tuberculous laryngitis. Because of the relative starvation caused by the extreme pain on taking food, and by the regurgitation of food through the nose.

6. Tuberculous enteritis. Because of the depleting effect of the prolonged diarrhea, and because of inability to digest or assimilate what little food can be taken.

7. Tuberculous meningitis. Because of the dullness stupor, and coma that ensue, making feeding al most impossible, because of the practically abso- lute constipation, and because of the spread of the tuberculous poison throughout the centra' nervous system.

Important complications of pulmonary tuberculosis.

1. *Hemoptysis* (hemorrhage from the lungs). Hemop- tysis is very frequent, occurring to a greater or less ex- tent in about 60 per cent. of patients. The hemorrhage may be of any size, from a teaspoonful to 2 quarts. It may be preceded by the expectoration of blood-streaked sputum, but more often it occurs suddenly and unex- pectedly. Hemoptysis is caused by the eating away or erosion by the tuberculous process of one of the pul- monary vessels. If the vessel is small, the hemorrhage is usually slight; if the vessel is large—*e.g.,* an artery run- ning across a cavity—the hemorrhage may be so great as to prove immediately fatal. Hemorrhages may occur singly, or several may follow one upon the other. At times, a hemorrhage may be directly traceable to some indiscretion—lifting, running, etc. More often, the ex- citing cause remains unknown.

Symptoms. Frequently there are none until the pa- tient expectorates a mouthful of blood. At other times a tickling sensation is felt under the sternum, a warm, salty taste appears in the mouth, and blood is expector- ated. The blood is usually bright red and frothy. It may come only on cough, which is apt to be very fre-

quent, it may come so fast as to almost choke the patient, or, in extreme cases, it may pour from the patient's mouth like water from a faucet. Later, blood is almost invariably vomited, as all is never expectorated, and some always trickles down the esophagus into the stomach. After a fair-sized hemorrhage, patients almost always run a higher temperature for a few days, due to the absorption of the blood that has remained in the lung.

Dangers of hemorrhage:

(*a*) Loss of blood.

(*b*) Aspiration pneumonia.

(*c*) Dissemination of tuberculous process.

But a small percentage of patients that bleed actually die from loss of blood. Occasionally a bronchopneumonia sets in, usually of tuberculous origin, from which recovery is rather the rule than the exception. If, as a result of hemorrhage, as not infrequently happens, a spread occurs in the area of disease in the lung, its extent and character will determine the fate of the patient.

2. *Tuberculous Laryngitis.* When we consider that every bit of germ-laden sputum that is expectorated passes through the larynx, the wonder is that cases of tuberculous laryngitis are not more frequent. The complication is, however, of very common occurrence. The first symptom may be any of the following:

(*a*) Weakness and rapid fatigue of the voice.

(*b*) Hoarseness in varying degree.

(*c*) Pain in the throat, usually on swallowing.

The amount of hoarseness and pain depend upon the extent of the process, and also upon the particular part of the larynx involved. Thus, a very slight involvement of the vocal cords will cause marked hoarseness, and sometimes complete aphonia, while far greater involve-

ment of other structures of the larynx will affect the voice slightly, if at all.

Where the epiglottis is involved there is very great pain on swallowing food, and food or fluid taken not infrequently regurgitates through the nose. At times the pain radiates to the ears. In advanced cases an enormous quantity of mucus is secreted, which has to be expectorated almost constantly, this serving to greatly exhaust the patient. In certain types of laryngeal tuberculosis the pain on trying to swallow food is so great that the patient literally prefers to starve to death.

3. *Intestinal Tuberculosis* (tuberculous enteritis). This condition may be primary—*i.e.*, the beginning of active tuberculosis in the affected individual. Usually, however, it is secondary to advanced pulmonary disease. It is not an uncommon complication, and is very serious indeed, practically all cases going steadily downhill. Ulceration occurs in the large and small intestine. Hemorrhage from the ulcers may occur, though this is rare as compared with hemorrhage from typhoid ulcers, because of the nature of the tuberculous process, which tends to block up and shut off the blood supply from the invaded area. Tuberculous ulcers have their long axis *around* the intestine, in contrast to typhoid ulcers, whose long axis runs *lengthwise* to the intestine.

The main symptom of intestinal tuberculosis is an *obstinate, intractable, painful diarrhea,* sometimes alternating with periods of constipation, the stools numbering from 4 to 12 per day, and having a rather characteristic and extremely offensive odor. With this diarrhea there is profuse gas formation, almost constant abdominal pain, a distaste for food, and marked and progressive emaciation. Death usually occurs from exhaustion.

4. *Tuberculous Meningitis.* This complication, though not as common in adults as the three preceding ones, is not infrequently met with. It is extremely fatal, many authorities placing the mortality at 100 per cent. There are no absolutely characteristic symptoms of tuberculous meningitis that serve to differentiate it from any other meningitis, save the examination of the spinal fluid (see section on Lumbar Puncture in chapter on Epidemic Cerebrospinal Meningitis) and the finding therein of tubercle bacilli.

When, however, a tuberculous patient presents the three following symptoms, the existence of a tuberculous meningitis becomes practically a certainty :

(*a*) Headache. Marked, persistent, becoming gradually worse. Resistant to all manner of treatment.

(*b*) Vomiting. Constant, not associated with the taking of food.

(*c*) Constipation. Marked, and growing progressively worse. Short of absolute intestinal obstruction, there is no more marked constipation than that found in tuberculous meningitis.

In addition there are present symptoms of meningitis in general—at first those of cerebral irritation, and later those of cerebral depression. (See chapter on Epidemic Cerebrospinal Meningitis). Patients die from exhaustion, starvation; from absorption of toxins from the bowels; and from the spread of the tuberculous process throughout the central nervous system.

Prophylaxis. The prevention of tuberculosis is the cornerstone upon which is erected the hope for the

future eradication of the disease. Prophylaxis may be divided into four classes:

(1) National.
(2) State.
(3) Municipal.
(4) Individual.

National and state prophylaxis do not come within the scope of these lectures. Municipal prophylaxis also will not be considered in detail, but the following list will serve to show the different paths by which control and prevention of the disease is being sought:

(a) Report to the local board of health of all cases of tuberculosis.
(b) Tuberculosis clinics.
(c) Tuberculosis classes.
(d) Day camps for the tuberculous.
(e) Night camps for the tuberculous.
(f) Sanatoria.
(1) For incipient cases.
(2) For advanced cases.
(g) Public lectures, free of charge.
(h) Public exhibits, free of charge.
(i) Posters illustrating preventive measures.
(j) Instruction leaflets widely circulated, free of charge.
(k) District tuberculosis nurses to visit the homes of the tuberculous poor, free of charge.
(l) Providing, free of charge, the few necessaries to make the tuberculous individual no longer a source of danger, and to enable him to take rational care of himself—sputum cup, thermometers, disinfectants, etc.

Individual Prophylaxis. This subdivision will be discussed more in detail, as the nurse must see that the necessary precautions are scrupulously carried out, both for her own protection and for that of the members of the family of her patient.

(*a*) *Care of the Sputum.* It is probable that at the end of fifty years "if all sputum were destroyed there would be no tuberculosis," for the germ-laden sputum is by far the most important agency in spreading the disease. Every patient having sputum, whether bacilli have been found in it or not, should possess a sputum-box, and invariably expectorate in that box. The best box is one consisting of a tin holder into which paper fillers are fitted. Every twenty-four hours the filler (whether full or not) is removed from the holder and burned with its contents. Very finicky patients will not, and very weak patients often can not, use a sputum-box. For such patients the nurse should provide small squares of cheesecloth, gauze, or tissue paper into which the sputum can be expectorated. An ordinary paper bag pinned to the sheet within easy reach of the patient serves as a receptacle for these cloths, and every twelve hours the bag and its contents should be burned.

(*b*) *Covering the Mouth when Coughing.* The nurse should provide a liberal number of gauze or cheesecloth squares for this purpose, as it has been shown that in the act of coughing minute particles of sputum containing bacilli may be expectorated. A nurse should insist upon the patient's observing this rule, which is the one above all others that even conscientious patients are prone to neglect.

(*c*) *Separate Dishes and Table Utensils.* The patient should use separate dishes from the rest of the family.

It is well for the nurse to suggest that these dishes be of a different pattern. Silverware (knives, forks, and spoons), napkins and tray-cloths should also not be mixed with the family's. Paper napkins are desirable, as they can be burnt.

(*d*) *Frequent Hand-washing.* The patient should wash the hands frequently, especially before and after meals, and should repeatedly rinse the mouth with some mildly antiseptic mouth wash, such as Dobell's solution.

(*e*) *Bedclothing and Bedlinen.* These should be dealt with separately from the family washing, and should be thoroughly boiled.

(*f*) *Care of Thermometers, etc.* All thermometers should be kept in a bichloride (1 : 1000) or carbolic (5 per cent.) solution, and washed with water before being given to the patient. Rectal tubes, enema nozzles, etc., should be sterilized after use by boiling.

(*g*) *The Nurse's Care of Herself.* The nurse should insist upon a reasonable amount of time off duty, should take a daily brisk walk of at least half an hour, should pay scrupulous attention to the care of her hands and mouth, and should never use any article that has been used by the patient.

By rigid adherence to the few simple rules here given, the patient will prove absolutely no danger to the household in which he lives, and the nurse will be doing, in addition to her professional duty, an educational work in the family and in the community in which she is called upon to practice.

Treatment. The treatment of tuberculosis may be divided into four groups:

    (1) Hygienic-dietetic treatment. By far the most important.

(2) Specific treatment, *i.e.*, tuberculin. ⎫ In con-
(3) Treatment by the induction of ar- ⎬ junction
    tificial pneumothrax. ⎭ with 1.
(4) Symptomatic treatment.

Hygienic-dietetic Treatment. This is based on three equally important factors:

    (*x*) Rest.
    (*y*) Fresh air.
    (*z*) Food.

The nurse will so rarely be called upon to care for the case that is truly ambulant that it is not necessary to go into the details of the régime. A few general rules will merely be laid down without comment:

    (1) Rest at first, and until exercise is ordered.
    (2) Day spent on porch in reclining chair.
    (3) Temperature and pulse taken four times daily and oftener at first.
    (4) Three full meals per day, at the usual hours, supplemented by such additional nourishment as ordered.
    (5) Exercise when ordered, and in the amount prescribed.
    (6) Sleep on porch or in room with all windows open.
    (7) In bed not later than 10 P.M.
    (8) Drugs only for combating individual symptoms.
    (9) No alcohol in any form.

Bed Cases. The vast majority of cases of pulmonary tuberculosis employing a trained nurse are bed cases at the time of the nurse's arrival. As a typical example will be selected a moderately advanced case, running a maximum daily temperature between 101° and 102°, with other symptoms in proportion.

I. *General Management.* If the patient has a sleeping porch it will, of course, be used. See that the bed is in a protected portion of the porch, and that the patient is not liable to be wet by a driving rain. If necessary, ask for an awning or a canvas shield for the exposed end of the porch. If there is no sleeping porch available, the room must be as freely open to the air as possible. Cold, rain, etc., are no contraindications to this, it being of course understood that the patient is to be at all times warm and comfortable. The head of the bed should not be in a corner where there is air stagnation, nor between two windows where a direct draught blows on the patient, but well out in the body of the room where air circulates freely. Sacrifice the looks of the room to the welfare of the patient. In winter the nurse must be sure to provide *herself* with warm clothes, both under and outer garments, in order that she, too, will be comfortable in cold weather. The writer has known of several nurses rendered seriously ill by the combination of insufficient clothing and devotion to their patients. The nurse owes her services to her patient, but she owes her health to herself; it is her most precious asset.

Save in conditions of great weakness, or after hemorrhage, the bedpan is usually not necessary. If the bathroom is convenient, it can be used, or else recourse can be had to a commode.

Care must be taken not to expose the patient in cold weather. If a porch is used, he must be rolled into the room for the morning toilet, which differs in no way from that of any case of febrile disease—bath, rub, etc. If the patient is in a room, *it must be warm* before any work is undertaken with the patient. This is excessively important, and often neglected.

II. *Food*. Tuberculosis being a wasting disease, food is excessively important. The normal caloric needs of the body must be exceeded, for oxydation of foodstuffs and of tissues is going on more rapidly than in the normal individual. There are no absolute rules for diet in tuberculosis. There is no one diet in tuberculosis. Generally speaking, the caloric needs of the patient will be supplied by "three square meals a day and a little more," the "little more" coming in the form usually of eggs and milk, to be taken as prescribed by the physician.

Certain general principles of diet will be mentioned. The application of accurate caloric feeding is rarely practiced, save in an institution under the supervision of a dietician assisted by several nurses. In the ordinary case encountered in private nursing a general estimate of the caloric value of food taken will be made, and feeding directed upon that as a basis.

Food for the tuberculous should be well prepared— cleanly, promptly, and attractively served. The ordinary articles of diet are satisfactory. Meat should not, as a rule, be eaten more than once a day. It is not wise to increase too greatly the proteid intake. For the sake of gaining weight, carbohydrates and fats should be increased more than the nitrogenous food. Extra nourishment is usually indicated in the majority of cases. The simplest way to give it is in the form of milk and eggs. Fortunately, the days of tremendous overfeeding are past, and now the object is to give just as much as the stomach will tolerate, but no more.

Many tuberculous patients, especially those running some temperature, have poor appetites, and a considerable part of the nurse's duty will be to try and make these patients eat. The few suggestions on the prepara-

tion and serving of food for the sick in the chapter on "Foods and Nutrition" apply particularly well to tuberculosis. Many patients announce at once: "I cannot take milk and eggs." As a matter of fact, this usually means: "I dislike milk and eggs, and I don't want to take them."

There are some patients that really *cannot* take milk and eggs, every attempt so to do causing marked symptoms of indigestion. These patients are greatly handicapped, but fortunately their number is small. As a rule, by coaxing, by disguising the taste of the milk with very little tea or coffee, by beating up the egg in the milk and adding a little vanilla, by having milk and egg ice cold, by beginning with the white of the egg and not adding the yolk until later, or by many other little subterfuges, the patient, if really in earnest and co-operative, can manage to take eggs and milk.

Too much care and attention cannot be expended by the nurse on the patient's food. The tripod upon which rests the treatment of tuberculosis is: rest, fresh air, food. Rest can be obtained; fresh air is within the reach of all; but food not only must be well selected but well cooked, and served in such a manner as to overcome aversion on the part of a stomach that instinctively revolts at the thought of a meal.

III. *Bowels*. The care of the bowels is extremely important. Save in those cases of tuberculous enteritis, or during some transitory intestinal derangement, constipation is the rule. It is very natural that this should be so, for the patient is put to bed, allowed no exercise whatsoever, and fed very liberally. Many of the cases of constipation clear up in a marked degree when the patient is able to take thirty minutes' exercise. Laxatives must

be resorted to in the majority of cases. As a general rule, it can be stated that it is better for the patient to have two bowel movements daily than to go one day without a thorough evacuation.

IV. *Cough.* There is no symptom more wearing and exhausting than cough, and many bed-patients are actually greatly overexercising as a result of the exertions incident to the cough. There are, generally speaking, two kinds of cough in pulmonary tuberculosis:

(1) Dry—hacking—bringing up no sputum. This cough, like that in the beginning of lobar pneumonia, is "never helpful, always troublesome, sometimes dangerous," and should be discouraged. About 75 per cent. of it can be controlled by the will. The nurse should keep this before the patient, and gradually she will see the fruits of her suggestion in lessened hacking, more rest, and increasing strength. At times, cold cloths or an ice-bag to the throat is of great value in relieving the dry, harassing cough. Almost invariably, some drugs are necessary to help the cough, an opium derivative, like codeine or heroin being usually the cornerstone of the prescription.

(2) Loose, productive cough, bringing up sputum. This type of cough is beneficial. It is Nature's method of drainage, and should not be interfered with.

V. *Temperature.* Rest in bed is the best treatment for fever. Moderate temperatures, up to 103°, rarely require any active treatment other than bed-rest. With higher temperatures, cold, in the form of the ice-cap,

gives relief, as do also sponges with alcohol and water. With very high fever, or in patients that feel very badly indeed with a moderate amount of temperature, antipyretics are used.

VI. *Night-sweats.* As night-sweats are simply a symptom of toxemia, that which reduces the toxemia will also cause the disappearance of the sweats. Rest in bed is the best treatment for night-sweats, as it removes, or at any rate lessens, the cause. Drugs are also of value for night-sweats, several being used, the most reliable being atropin and camphoric acid. Alcohol and vinegar rubs at night are also sometimes of benefit.

VII. *Insomnia.* Often very intractable. The success of its management depends almost entirely upon the underlying cause. If cough is the cause, its alleviation will be of great benefit. For the sleeplessness apparently without cause that so often troubles tuberculous patients, but little is to be done. The condition is probably an expression of toxemia, and rest in bed is the best treatment. Practically all physicians hesitate to give hypnotics in these cases because of the great dependence so soon placed upon them, but often it is absolutely necessary to employ them for a short while.

VIII. *Vomiting.* There are two kinds of vomiting seen in tuberculosis:

(1) Vomiting due to local stomach conditions. The digestive system is then at fault and treatment must be directed toward the correction of whatever is out of gear.

(2) Vomiting due to coughing, and of purely mechanical origin, there being no disturbance whatsoever of the gastro-intestinal tract. This vomiting is particularly marked during or after breakfast. The warm food and

coffee taken at breakfast serve to loosen the secretions in the lungs. These cause cough in order that expectoration may take place. The diaphragm pressing down with each cough upon the recently filled stomach, finally causes a gastric contraction which results in vomiting. These cases can often be very well dealt with by giving the patient a glass of hot water on awakening. The water is to be sipped slowly. It acts as a "loosener" to the secretions, and coughing and expectoration take place before breakfast, and on an empty rather than on a full stomach.

Management of important complications.

1. *Hemorrhage.* The following facts must be plainly understood with regard to pulmonary hemorrhage:

(1) Hemorrhages are largely self-limited.

(2) No treatment by drugs for *rapidly* stopping hemorrhage is of much avail unless instituted within five minutes after bleeding has begun.

(3) Certain symptoms that make for more free bleeding can be satisfactorily controlled by drugs.

(4) The mental attitude of the patient during a hemorrhage is as important as anything connected with the treatment of the condition.

(5) The attitude of the nurse in an emergency such as hemorrhage will largely determine the attitude of the patient.

The patient, with very few exceptions, is badly frightened. The nurse must keep her head, be calm, take charge of things, and convey the impression that bleeding is nothing over which to be alarmed. Her place is with her patient—not calling up half-a-dozen telephone

numbers in vain attempts to locate the physician. That important duty should be delegated to someone else.

That we have no specific for pulmonary hemorrhage is shown by the fact that almost every drug in the pharmacopeia has been used at some time or other. This, too, is a strong argument in favor of the self-limiting nature of pulmonary hemorrhage. All the drugs used cannot be mentioned. A brief statement of the general management of hemoptysis, and a few words concerning some of the most used methods will suffice.

The patient that is bleeding should at once be put to bed, if not already there; one pillow under the head (some authorities preferring an almost erect position), small amounts of salt and cracked ice by mouth. The patient should not be allowed to raise himself on his elbow to expectorate into the sputum cup. Sputum should be received into cloths—gauze, towels—anything that is at hand, and as far as possible the position of the patient should not be disturbed.

As to drug treatment, morphia is very often and very freely given—sometimes too freely. Morphia is, of course, the great drug for allaying intense nervousness and uncontrollable cough, and in a large percentage of cases will be indicated and required. For frequent distressing cough, codein acts very well, as does heroin, both being given hypodermically. The drug that has given the author the best results in the control of bleeding is atropine, $\frac{1}{33}$ to $\frac{1}{25}$ grain hypodermically. The dose is large, but the effect is to reduce deep blood-pressure by the dilation of the superficial vessels all over the body. The results are prompt, if administered at once upon the appearance of free bleeding. An amyl nitrite pearl is frequently given the patient while the hypoder-

mic is being prepared. Emetin hypodermically is now frequently employed, as is also pituitrin.

The calcium salts are frequently used in hemorrhage cases, because of their action in increasing the coagulability of the blood. The chloride and lactate of calcium are the salts employed. In cases of multiple hemorrhages, or prolonged oozing, horse serum, ergot, adrenalin, coagulose and thromboplastin are among the agents that have been advised. The writer has seen good results from the administration of coagulose.

After a hemorrhage, the patient should be kept on his back in bed until the sputum is again clear. For several days after active bleeding there is sure to be red sputum, and the nurse must reassure the patient that this does not signify continuance or renewal of bleeding.

For twenty-four hours after a smart hemorrhage nourishment should be liquid, and nothing hot should be given until the sputum is again clear. Attention must be given to keeping the bowels well open by laxatives or enemata.

2. *Pleurisy.* This subject is dealt with in the chapter on "Pleurisy—Dry and with Effusion," and therefore will not be discussed here.

3. *Tuberculous laryngitis.* The actual treatment of tuberculous laryngitis falls outside the province of the nurse. There are a few things, however, that she can do for patients with this complication:

(*a*) Spraying the throat. Sprays or powders, both for treatment and as anesthetics, are often prescribed, and rarely satisfactorily administered. A spray or powder improperly given is worse than no spray at all. Hence, the following directions for spraying the larynx are given:

(1) Turn adjustable tip of atomizer downward until it makes an angle just *short* of a right-angle.

(2) Let patient sit upright facing a good light, either natural or artificial.

(3) Let patient pull out tongue as far as possible with a piece of gauze, and hold it thus. (This raises and immobilizes the larynx.)

(4) Quickly insert barrel of atomizer into mouth, holding it in the median line, and having the tip about ¼ inch from the posterior pharyngeal wall.

(5) Tell the patient to take a long, slow breath, and during that breath press bulb of atomizer vigorously three or four times.

(6) As soon as patient begins to gag, withdraw atomizer, as its contents can no longer reach their goal.

(7) Repeat this three or four times at the specified hour at which the spray is used.

(8) For the insufflation of powders the procedure is exactly the same, save that two good "puffs" of the powder are usually enough for one dose.

(b) Cold to the throat. This should be applied either by cold cloths constantly changed, or by means of the throat ice-bag, which adapts itself to the shape of the neck. The ordinary ice-bag or ice-cap is useless for this purpose.

(c) Fly-blisters (cantharides plasters) are often used on the sides of the neck over the point of maximum laryngeal pain.

(*d*) Silence. If talking is prohibited, the nurse must see that silence is enforced. She must have paper and pencil at hand for the patient to write upon, and she must never answer any spoken question.

4. *Tuberculous Enteritis.* Unfortunately, there is very little to be done for this distressing condition, and it is usually one of the terminal phases of pulmonary tuberculosis. Opium in some form, usually laudanum, must be prescribed to check diarrhea and to lessen pain.

5. *Tuberculous Meningitis.* The treatment of this complication is purely symptomatic. At times, much relief can be obtained by frequent lumbar punctures, which, by lessening pressure in the spinal canal and in the ventricles of the brain, often causes great relief in symptoms, this relief being, unfortunately, only temporary.

Tuberculin Treatment. It is not intended in these lectures to touch upon the question of treatment with tuberculin, for that rests wholly within the province of the physician. The following statements can be made, however.

1. Tuberculin is any substance derived directly or indirectly from the tubercle bacillus and used therapeutically.

2. There are over fifty varieties of tuberculin.

3. The object of treatment with any tuberculin is to stimulate the body to the greater production of protective substances (antibodies) against the tubercle bacillus and its toxins—*i.e.,* the bringing about of an active immunity to tuberculosis.

4. There is no doubt that in certain cases tuberculin can be of inestimable value.

5. There is no doubt that in the past tuberculin has been held up as a poison which it was criminal to use, and has been given credit for working miracles. Both extreme positions are unjustifiable.

Treatment by the Induction of Artificial Pneumothorax. This mode of treatment, first devised by Forlanini, of the University of Pavia, Italy, in 1882, merits a short consideration. The object of artificial pneumothorax is to collapse and immobilize the affected lung by means of an "air splint," and, as a result of this collapse and immobilization, to further healing and scar formation by giving absolute rest to the diseased organ.

In the small operation (which corresponds very much to tapping the chest for fluid) necessary for the induction of artificial pneumothorax, a blunt, hollow needle is inserted between the ribs until its point is between the two pleural layers, this being indicated by certain characteristic fluctuations of a column of water in a U-tube, known as a manometer, which is connected by a tube with the needle in the chest. The point of the needle being in the desired position, the manometer is turned off and nitrogen gas or sterile air allowed to flow in. The gas spreads, of course, in the direction of least resistance. Toward the outside are the ribs and the firm intercostal muscles, forming an unyielding wall. Toward the inside is the soft, spongy lung, which gives way and shrinks much as does a sponge when squeezed. Gradually, after several injections, the lung is completely collapsed, the entire pleural cavity being filled with gas. When successful, the collapse of the diseased lung causes a prompt diminution in all symptoms, a lessening of fever, cough, sputum, a return of strength and well-being that in some cases is little short of miraculous.

Collapse is maintained for from six months to three years, and at the end of that time, healing having taken place, no more gas is given, and the lung slowly re-expands.

Two factors are necessary for the induction of artificial pneumothorax:

1. One sound or almost sound lung, in order that it will, unaided, be able to carry on the task of respiration.

2. Absence or scarcity of pleural adhesions. If adhesions between the two pleural layers are so dense that they will not give way under pressure from the gas, no collapse can be obtained and the procedure cannot be used. Failure occurs in about 33 per cent. of all attempts.

Thus, taking the treatment of pulmonary tuberculosis and very briefly summarizing it, the following scheme can be presented:

1. Hygienic-dietetic treatment. Applicable to every case. Based essentially on

    (*a*) Rest.
    (*b*) Fresh air.
    (*c*) Food.

2. Treatment with tuberculin. Applicable to a moderate number of cases.

3. Treatment by means of artificial pneumothorax. Applicable to a very small percentage of cases (about 5 per cent.).

4. Symptomatic treatment. Very important, and, in conjunction with No. 1, applicable to every case.

CHAPTER XXIII.

SCARLET FEVER.

SCARLET fever is an acute infectious disease having as its main features a characteristic rash, an inflamed and painful throat, and a high temperature, usually of short duration. One attack of scarlet fever usually confers immunity from further attacks, though this rule has occasional exceptions. It usually occurs before the tenth year, but infants are rarely attacked.

The disease is very highly contagious, and is looked upon as being probably an infection with some form of streptococcus. The main sources of contagion are:

1. The patient.

2. The room occupied by the patient and its contents.

3. A third person—nurse or doctor. This mode of contagion must be very rare, and is wholly denied by some authorities.

The period of incubation			is from		2 to	6 days.
"	"	" invasion	"	"	12 "	24 hours.
"	"	" eruption	"	"	4 "	6 days.
"	"	" desquamation	"	"	3 "	6 weeks.

Symptoms (average attack). The onset is sudden, and often accompanied by vomiting. The temperature rises rapidly to 103° or 104°, with the usual symptoms of fever. There is redness of the pharynx and tonsils, and small red spots are seen on the hard palate. The redness of the throat is somewhat characteristic. It is a dark, deep crimson blush, quite uniformly spread over the entire pharynx and both tonsils. The tongue is known as

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the "strawberry" tongue, and is very well named. The papillæ at the tip of the tongue are swollen, and the resemblance to the rough surface of a strawberry is very marked. The glands of the neck àre invariably swollen and tender.

The rash develops in from twelve to thirty-six hours after the appearance of the first symptoms. It is first seen on the neck and chest, and gradually spreads over the entire body. The rash is what is known as a uniform diffuse erythema. On close examination it appears to be made up of countless minute red points. When developed, it gives the impression of an evenly distributed blush, not distorting the countenance, as does the rash of measles. Unfortunately, the rash of scarlet fever often varies, both in character, intensity, and distribution, and at times may be almost absent. Such cases, with very slight rash, are often unrecognized, and must be one of the important factors in furthering the spread of the disease. The rash lasts from three to seven days, when desquamation sets it. With subsidence of the rash the temperature gradually drops to normal.

Desquamation in scarlet fever is a very long process, lasting from three to six weeks. Peeling takes place in the form of very fine scales, and is most apparent in those portions of the body where the skin is thickest—*i.e.,* the palms of the hands and the soles of the feet. In these localities desquamation often occurs in large "sheets," occasionally an entire "cast" of the palm of the hand or the sole of the foot being given off.

In mild cases the temperature may not exceed 103°, and lasts from three to five days. In severe cases, the fever is higher and more continued, there is greater prostration, and all the signs of a severe general infection are marked.

Finally, there are the cases of so-called "malignant" scarlet fever, in which the patient is completely overwhelmed by the intensity of the infection, and death occurs in from twelve to seventy-two hours.

Complications.

1. Acute nephritis. Scarlet fever is one of the most common causes of acute nephritis. The symptoms of this condition are dwelt upon in detail in the proper chapter, but a few words must be said here concerning this dangerous complication. The nurse must remember that the two signs of a beginning nephritis in the course of scarlet fever are:

(*a*) Edema. The child has a rather puffy look, especially noticeable in the face.

(*b*) Diminution in the amount of urine.

Either one or both of these symptoms are of the utmost importance, and demand immediate notification of the physician.

The nurse must also bear in mind two things in connection with scarlatinal nephritis:

(1) The most severe nephritis may occur as a complication of the mildest attack of scarlet fever.

(2) Acute nephritis very frequently occurs during convalescence from scarlet fever.

In short, no nurse caring for a case of scarlet fever can lower her vigilance for the signs of a beginning nephritis until she is dismissed from the case. Usually, the patient's condition during the long period of desquamation is so satisfactory that daily visits on the part of the physician are not needful, when he is aided by the watchfulness of a competent and interested nurse.

2. Acute otitis media. This is the most frequent complication of scarlet fever, but is not as dangerous as acute nephritis. If the otitis occurs at the height of the disease, there may be no symptoms. If during convalescence, earache and a rise in temperature are characteristic. As a rule, both ears are involved at different times.

3. A membranous inflammation of the larynx may occur, giving rise to symptoms similar to those observed in laryngeal diphtheria (*q.v.*).

4. Other infectious diseases, especially diphtheria, may complicate scarlet fever.

Prognosis. The outlook in scarlet fever is always serious. The younger the child the graver the situation. Save for the malignant cases, scarlet fever, in the absence of complications, is not a very fatal disease, but the frequent occurrence of dangerous complications makes it a malady to be dreaded. In mild types of the disease the mortality is under 5 per cent. In severe types, it may be as high as 50 per cent.

Prophylaxis. The following directions for the establishment of quarantine hold good for scarlet fever, measles, and diphtheria, varying only in the length of time quarantine is to be maintained.

Room quarantine is to be installed at once, and maintained for the time designated by the local board of health—as a rule, from four to six weeks. Nurse and patient are to be isolated in the sick-room. When possible, a connecting bath is desirable, and, if practicable, a little diet kitchen should be installed in the bath-room, using a small gasoline or gas stove, so that no article need be sent out of the sick-room. In the vast majority of cases of scarlet fever, such conveniences will not be obtainable, and the best possible must then be done.

A sheet moistened with bichloride of mercury solution (1 : 1000) or carbolic acid (5 per cent.) should be hung before the door. In the author's opinion, this procedure has no great value in preventing the dissemination of infectious material, but the striking appearance of the sheet before the door has a wholesome effect upon members of the family inclined to carelessness, and acts admirably as a "No Admittance" sign.

All food brought to the sick-room should be left at the door, and taken in by the nurse. Before the dishes and other utensils are replaced outside the sick-room they should be allowed to soak for two hours in bichloride (1 : 1000) or carbolic (5 per cent.) solution. All bed linen, towels, gowns, etc., should be similarly treated before being set outside to be washed. Both dishes and bedclothes should not be washed in conjunction with those used by the family.

The room should be cleared of all unnecessary furniture, rugs, curtains, etc., and should be frequently cleaned by the nurse by being rubbed with a cloth wet with bichloride (1 : 1000).

The attending physician should have a gown and cap which he should put on every time he visits the patient. These should be hung on a hook just inside the door. If no gown and cap are available, a very satisfactory gown can be made from a sheet, and a small towel pinned "turban fashion" makes a thoroughly practical cap. The nurse should have at hand a basin with soap and water, a basin of bichloride solution, and a towel for the physician at his visit. She should also request tongue depressors (wooden ones are the best), so that throat examinations can be easily made, and recourse to the unhygienic spoon become unnecessary. If the nurse will

provide herself with a pocket flash-light, which she will find useful in many ways, she will often greatly aid the physician, who may have left his at home.

At the termination of the period of quarantine the nurse should give her patient a bath in bichloride (1 : 5000), and wash the hair well with this solution. Following this, the patient should have an ordinary hot bath, and put on clothes that have not been in the sick-room. The nurse should then take her own bichloride bath, wash her hair, take a hot bath, and also put on clothes that have not been in the sick-room.

Treatment. We have at our command absolutely no means of shortening or altering the course of scarlet fever, which is wholly a self-limited disease. Treatment is purely symptomatic.

Bed, liquid diet during the period of fever, and keeping the bowels well open, are the foundation-stones for the management of a case of scarlet fever. During the eruption the patient should be anointed daily with vaseline or cocoa butter. After the rash has disappeared, daily warm baths with soap and water are frequently used. For very high temperature cold sponging gives the best results. When toxemia is very severe, stimulation may be necessary, according to the discretion of the physician in charge. It is usual to give a gargle or to prescribe some antiseptic with which the throat is to be swabbed.

One of the essentials in nursing scarlet fever is eternal watchfulness for complications. During the long and tedious period of desquamation, when the child feels quite well, and must still remain isolated, the nurse will have to tax to their uttermost her talents for diverting and amusing her little patient.

CHAPTER XXIV.

MEASLES.

MEASLES is an epidemic contagious disease, and is more widespread than any other eruptive fever.

> *Incubation, i.e.,* from date of exposure to onset of catarrhal symptoms, from eleven to fourteen days.
>
> *Invasion, i.e.,* from onset of catarrhal symptoms to the development of the rash, usually three to four days.
>
> *Eruption, i.e.,* duration of the rash, four to six days.
>
> *Desquamation, i.e.,* "peeling," one to two weeks.

Etiology. The essential cause is unknown, though it is believed to be some germ as yet not isolated. Only a short exposure is necessary for its communication, and close proximity to the infected individual is not necessary. The disease is highly contagious from the onset of the catarrhal symptoms, and, as the patient is not ill and confined to the house at that time, measles is spread on all sides. After the disappearance of the rash and catarrhal symptoms, the communicability rapidly decreases, and during desquamation is but slight. Generally speaking, the duration of the infective period is three weeks.

Children are usually attacked. Very young infants are not as susceptible to measles as those somewhat older, but in the very young the disease is a serious matter. The vast majority of those having measles are under 12 years of age. One attack usually produces an

immunity to measles, but there are many exceptions to this rule, and two, and even three, attacks are not very uncommon.

Symptoms (average attack). The disease is ushered in with symptoms of a diffuse catarrh of the upper respiratory tract. The patient has a "running" nose, "running" eyes, a sore throat, with redness of the tonsils, and soft palate, a hoarse, harsh cough, and, in a day or so, some sputum. The catarrhal process spreads to the bronchial tubes, and bronchitis is present so frequently as to be looked upon as a symptom of measles, and not as a complication.

The temperature rises gradually until the appearance of the rash, reaches about 104° as a maximum, and lasts in all usually about a week, varying from five to nine days. At first there is some dullness, pain in the back, headache, and general malaise, feelings that accompany any moderate rise of temperature, and that present no characteristic features. Vomiting and diarrhea are rarely seen save in the severer forms.

Before the appearance of the rash there is but one sign that will point without question of doubt to measles. This sign is *Koplik's spots*—bluish-gray spots seen against a red background on the mucus membrane of the cheeks and lips.

The rash of measles appears, as a rule, on the fourth day of the disease. It is known as a maculo-papular rash. It appears first behind the ears, around the neck, and at the roots of the hair, as small, dark-red spots, not numerous, not elevated, and looking somewhat like flea-bites. In twenty-four hours the macules are numerous, and many have become papules. The rash spreads rapidly to the chest, arms, trunk, and eventually involves

the whole body within thirty-six hours. The papules, which have at first been single, may fuse, and, in so doing, often assume a crescentic form. At the height of the disease the patient may be so disfigured by the rash as to be unrecognizable.

The skin is swollen, there is great itching, the eyes are red and very sensitive to light, and, as a rule, there is a conjunctivitis, with the formation of mucopus. Pain on swallowing, and swelling of the cervical glands are common. With the fading of the rash the temperature drops gradually, and reaches normal in from two to three days.

Also, with the fading of the rash, desquamation or peeling sets in. This is first noticed on the face and neck and is in the form of fine, branny scales—never in large patches, as is the case in scarlet fever. As mentioned before, desquamation lasts from one to two weeks—usually about ten days.

Some cases of measles are so mild that were it not for other cases in the family or immediate neighborhood they could not be recognized. On the other hand, other cases are so severe that either the patient is overcome by the systemic poison within a few days, or else the whole force of the infection seems to be expended upon the lungs, and the case is more one of bronchopneumonia than measles. Some severe cases have a hemorrhagic rash; others have convulsions and delirium, with all the signs of intense general poisoning.

Complications.

1. Bronchopneumonia. Frequent and dangerous. The symptoms that will cause the nurse to suspect a bronchopneumonia are:

(a) Rise in temperature.

(b) Rise in pulse-rate.

(c) Rise in rate of respiration.

(d) Increase in cough, and, in older children, in expectoration.

(e) Appearance of slight cyanosis in young children or in the very delicate.

2. Otitis media. Also frequent, but not as dangerous as bronchopneumonia.

Older children will usually complain of pain in the ear, and thus the nature of the trouble can be suspected, but in very young children and in infants the nurse must be constantly on guard for some change in her patient that will make her suspect otitis media.

In the very young the following symptoms are suggestive:

(a) Rise in temperature not traceable to the bowels or lungs.

(b) Fretful and persistent crying.

(c) Difficulty in taking the bottle (in infants).

(d) *At times* evident pain and tenderness in the region of the ear.

With such symptoms the nurse should at once call the physician, who will make the diagnosis by examination of the ear through an ear-speculum.

Diphtheria and scarlet fever may complicate measles. Kidney complications (nephritis) are rare, as are heart affections. Laryngitis is present in practically every case. When membranous laryngitis occurs it is caused either by the diphtheria bacillus or the streptococcus, and the symptoms are those described under "Laryngeal Diphtheria."

Prognosis. The outlook in the better class of private practice is generally good in children over 3 years of age. In those younger, mortality is fairly high. In those over 3, the average mortality is from 4 to 6 per cent., and often it does not reach these figures.

In institutions the picture is reversed, largely because the patients come from the poorer walks of life, are underfed, and have poor resistance. Here measles plays great havoc, in some institutional epidemics the mortality ranging from 15 to 35 per cent.

Prophylaxis. Room quarantine is required by law for a variable period depending upon the ruling of the local board of health. As the details for maintaining this quarantine are the same (save in point of time) as for scarlet fever and diphtheria, they have been given but once, and will be found in the chapter on Scarlet Fever.

Treatment. Measles is a self-limited disease, and we have no means at our command to shorten or modify it. Treatment is, therefore, wholly symptomatic.

The room should be darkened (especially in summer) by means of blinds or green shades, and the electric bulb or lamp covered with a red shade, in order to lessen all possible irritation of the eyes. An initial purge with calomel followed by a saline or castor oil is usually given. If the eyes are painful, ice-cold cloths frequently give relief, and the mucopus appearing as a result of the conjunctivitis should be wiped away with small bits of old linen moistened in a solution of boric acid. Vaseline may be freely applied to the lids. Vaseline or cocoa butter should be rubbed over the child's entire body in order to allay itching.

The diet should at first be liquid. Later in the disease eggs, toast, cereals, gruels, ice cream and crackers may be added. After the appearance of the rash a daily warm bath should be given in addition to the inunction above referred to.

The cough will usually need some treatment. Generally, opium in some form is given, either as codeine or heroin, combined with an expectorant mixture.

In cases of excessively high fever (105° or over) recourse is usually had to cold sponges, with alcohol 1 part, water 3 parts. With failing heart, stimulation is indicated, though there is rarely need for this save in the presence of bronchopneumonia, when the treatment becomes that of the complication rather than that of measles. The eyes must not be subjected to any undue strain for several weeks after measles, and during and after convalescence the child must be carefully watched, and every precaution taken against "catching cold," for the mucous membrane of the entire respiratory tract is in a condition of lowered resistance, and is particularly susceptible to all manner of infection.

If cough continues for any considerable length of time after recovery from measles, the possibility of tuberculosis must be borne in mind, this disease being one of the most frequent sequels of measles.

CHAPTER XXV.

DIPHTHERIA.

DIPHTHERIA is "an acute infectious contagious disease characterized by the formation of a gray-white membrane on the tonsils, uvula, and soft palate, and by constitutional symptoms of varying intensity." .

Etiology. The Klebs-Loeffler bacillus discovered in 1883. Diphtheria is very contagious. Short exposure is all that is necessary for infection to take place, and in addition the disease is spread by "carriers"—*i.e.,* persons having virulent diphtheria bacilli in their throats, but because of a natural or acquired immunity, not ill with the disease.

Symptoms. The incubation period of diphtheria is from twelve hours to three days. While diphtheria may occur on any mucous membrane where the bacilli lodge and develop (nose, vagina, stomach, etc.), it is very rare to see the disease anywhere save in the pharynx or larynx, and these two forms only will be considered here.

1. *Pharyngeal Diphtheria.* The onset is reasonably sudden, with chilliness, headache, fever, not particularly marked. It is uncommon to see a temperature over 102°.

Within twenty-four hours the throat becomes sore. At first it is red, but soon spots of gray or dirty white appear on one or both tonsils. These increase in number, unite, and spread to the uvula and soft palate. In a fully developed case the back of the mouth is often seen to present an arch of grey membrane, reaching

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from tonsil to tonsil. The membrane is thick and tenacious, and when pulled off leaves a raw, bleeding surface. As the disease progresses, prostration becomes more marked, and signs of heart weakness are frequent. The membrane may disappear as a result of treatment, or, in unfavorable cases, may spread to the nose or larynx. Cultures taken from the throat show the presence of diphtheria bacilli in large numbers. The course of diphtheria is variable, lasting from six days to three weeks, but has been wholly changed since the introduction of treatment by antitoxin.

2. *Laryngeal Diphtheria.* The general symptoms are the same as those of pharyngeal diphtheria, save that, as a rule, prostration is more pronounced.

The first local symptom is a hoarse, brassy cough. The voice may be merely husky, or the patient may not be able to speak above a whisper. If the membrane continues to spread over the larynx, dyspnea sets in, due to obstruction to the free passage of air. Cyanosis sets in, slight at first, but, in severe cases, gradually increasing until the entire face looks dusky, the patient gasps for every breath, the pulse is rapid, small and weak, and the entire body covered with a cold sweat. In untreated or very virulent cases the larynx may be entirely filled by the membrane, and death from suffocation result.

Complications.

1. Nephritis. Almost a constant occurrence, and due to the action on the kidney of the toxin of the diphtheria bacillus. Usually transitory; not serious; and diagnosed by the urinary findings.

2. Cervical adenitis. The glands of the neck are very frequently involved. They are often swollen and tender. Occasionally they break down and suppurate.

3. Bronchopneumonia. This complication is always serious. The gravity depends upon the age of the patient (if a child, the younger it is the more serious is the complication) and the severity of the diphtheritic attack. Bronchopneumonia is particularly apt to complicate cases of laryngeal diphtheria. For symptoms of this condition, see chapter on Bronchopneumonia.

4. Various paralyses. Very important. Due to a definite toxic action of the diphtheria poison upon the nervous system. Many varieties of paralysis may occur, chief among them being:

     (a) Palatal paralysis (soft palate), causing a nasal voice.

     (b) Paralysis of any of the eye-muscles.

     (c) Paralysis of any of the accessory muscles of respiration; if at all extensive, this is characterized by a peculiar sighing respiration.

5. Heart-failure. Most important of all. The toxin of the diphtheria bacillus has a very definite selective action upon the heart-muscle, causing a degeneration of the muscle fibres—a toxic myocarditis. Myocarditis is suspected from the rate and quality of the pulse, and from the fact that any physical exertion has a marked effect upon the circulation. Neither extreme of pulse-rate is of good omen, for "a rapid pulse-rate is always cause for alarm, and a slow pulse-rate an indication of serious trouble."

6. Vomiting. When this occurs early in the disease it may be due to the temperature and malaise that accompany any acute infectious disease. When vomiting occurs late in the disease, it is a very important and very dangerous symptom, as it points to beginning degeneration of the vagus nerve.

Prognosis. The outlook in diphtheria is always grave, though its course and termination have been so entirely revolutionized by treatment with antitoxin that this factor must always be held in the foreground. The gravity of prognosis and the rate of mortality are in direct proportion to the *delay* in administering antitoxin.

Kossel has shown that when antitoxin is injected on the first sign of the disease the percentage of recoveries is 100. In this every hour counts.

Out of 2428 cases reported by Hilbert, the percentage of deaths varied with the day on which antitoxin was administered, as follows:

Day of administration		Mortality	
First day	....	2.2 per cent.	
Second "	....	7.6 "	"
Third "	....	17.1 "	"
Fourth "	....	23.8 "	"
Fifth "	....	33.9 "	"
Sixth "	....	34.1 "	"
After sixth day	....	38.2 "	"

(Vaughan.)

The prognosis is always much graver in laryngeal than in pharyngeal cases.

Treatment. The treatment of diphtheria can be divided into two classes:

(1) General treatment.

(2) Specific treatment—*i.e.,* antitoxin.

1. *General Treatment.* (*a*) *Prophylactic.* The patient is to be isolated, as described in the chapter on Scarlet Fever, and quarantine is to be maintained until release is permitted by the board of health, the period varying in different communities, but being in all cases dependent upon cultures from the throat of patient and nurse showing no diphtheria bacilli.

(*b*) *General Management.*   Bed is to be insisted upon in all cases, and rest in the recumbent position is very important, owing to the toxic action of the diphtheritic poison on the heart.   The nurse must be careful not to let the patient sit up suddenly (as children are apt to do), as cases are on record where such exertion has caused sudden giving out of the heart, with immediate death.   The bowels are to be kept open, and the mouth clean with some mild antiseptic wash.   Gargling is not advisable, as it practically always necessitates sitting up.

Food should be liquid and semi-solid, bland, easily digestible, and given frequently in small amounts. Swallowing is at first usually very painful, and it may be very difficult to get patients, especially children, to eat a sufficient amount.   In toxic cases, stimulation is to be resorted to, differing in no wise from that given in any disease complicated by heart-weakness.

For the paralyses following diphtheria, strychnia seems to exert a more beneficial effect than any other drug.   The patient should be kept in bed until convalescence is fully established, because of the danger of sudden heart-failure even after all signs of active diphtheritic disease have disappeared.   Cold to the throat in the form of cold cloths is often beneficial.   The ice-bag is often very efficient in relieving pain.   Opiates may have to be given.

2. *Specific Treatment.*   There is probably no more brilliant achievement in internal medicine than the triumph gained over diphtheria by the discovery of diphtheria antitoxin by von Behring in 1890.

Antitoxin.   Antitoxin is, obtained by injecting horses with gradually increasing doses of diphtheria toxin,

until an immunity has been established, so that the
animal can withstand, with no harmful effects whatso-
ever, doses that would have proved immediately fatal if
given at first. When the horse is sufficiently immunized
he is bled, and in the blood-serum is found the diphtheria
antitoxin—*i.e.,* a substance which is capable of neutral-
izing and rendering harmless the diphtheritic poison cir-
culating in the patient's blood. The measure adopted.
for estimating the amount of antitoxin is the "unit."
The serum of the horse is standardized, and put up in
syringes ready for use, each holding a certain number of
units of antitoxin—500, 1000, 2000, 5000, 10,000, as the
case may be. Diphtheria being strictly a toxic disease
(*i.e.,* the poison manufactured by the diphtheria bacilli
being the one harmful factor), if enough antitoxin
is injected to neutralize all the toxin circulating in
the blood, the patient has an excellent chance for
recovery.

(*a*) *Prophylactic Treatment with Antitoxin.* It is
customary to give the nurse and all members of the
family a preventive injection of antitoxin—from 500 to
1000 units, according to the age of the individual to
whom it is given. This prophylactic injection estab-
lishes an immunity to diphtheria which lasts about three
weeks.

(*b*) *Active Treatment with Antitoxin.* Antitoxin
should be administered as soon as the diagnosis is made,
and, in doubtful cases, in the absence of a positive diag-
nosis, because if the case is one of diphtheria it will do
good, while if the case is one of follicular tonsillitis, no
harm will result, and because mortality from diphtheria
increases in direct proportion to delay in the adminis-
tration of antitoxin.

Antitoxin is administered hypodermically, the loose tissue of the back below the angle of the scapula being the favorite site of injection. The dose depends upon the judgment of the physician. Some believe in relatively small doses (2000 to 5000 units), others in moderately large doses (10,000 to 30,000 units), and still others in enormous doses (50,000 to 100,000 units). From this it will be apparent that, practically speaking, there is no such thing as an overdose of antitoxin. If the symptoms do not improve after the first injection, another is given from eight to twelve hours later, and subsequent doses are given as indicated. Where antitoxin has a beneficial effect, in from six to eighteen hours the membrane is seen to grow less, and finally to disappear; the temperature drops, and all signs of toxemia are reduced in intensity, the throat being often almost normal within three or four days. In more severe cases, a longer time in necessary for recovery; and in the fulminant cases, or in those in which antitoxin administration has been delayed until the entire body is flooded with poison, death ensues.

In cases of laryngeal diphtheria, in addition to antitoxin administration (always in larger dosage than in pharyngeal cases), mechanical means may be necessary to relieve the blocking up of the larynx, and consequent death of the patient from suffocation. These are two in number:

1. Intubation. By means of a special instrument for its insertion, devised by the late Dr. Joseph O'Dwyer, of New York, a hollow tube is passed into the larynx until it is between the vocal cords, where it lodges, and is held in place by means of a groove at its upper end. The patient is able to breathe through the tube until the mem-

brane lessens in amount and the larynx is again clear, when the tube is extracted if the patient fails to cough it out.

2. Tracheotomy. If intubation is impracticable, an opening is made in the trachea, and a tracheotomy tube inserted, through which the patient can get air.

CHAPTER XXVI.

ANTERIOR POLIOMYELITIS (INFANTILE PARALYSIS).

Definition. An acute infectious disease occurring both in epidemics and sporadically, due to a filtrable and cultivable virus, involving different parts of the nervous system, often localizing especially in the anterior horns of the gray matter of the spinal cord (poliomyelitis anterior), but also localizing in the cerebrum, in the medulla oblongata, in the cerebellum, and in the meninges, to a variable extent in different cases. Since the epidemic of poliomyelitis in New York in 1916, the medical thought of the country has been markedly concentrated upon this disease.

Etiology. After many experiments it has been established that the juices of the nervous system of an infected animal when filtered through porcelain or some other filter, are still able to infect monkeys. Hence the disease is due to a *filtrable virus*. This virus is most concentrated in the nervous system of the patient, but is also to be found in the mesenteric glands, and in the tonsils and throat. "The virus stands cold well, retaining its virulence when kept frozen for at least 11 days. It is enfeebled by a temperature of 45° C., and is killed after heating for half an hour at 55° C. It is not killed by drying. It can live for some time in sterile water or sterile milk, apparently without multiplication" (Barker). The transmission of the infection by the stable-fly, insects, fomites, etc., is still a debatable point. The

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disease is present chiefly in mid-summer and in the fall, though cases occur at all seasons of the year. The vast majority of those attacked are children from 1 to 4 years of age.

Pathology. Formerly it was believed that destruction of the anterior horn cells of the spinal cord was the sole characteristic lesion of poliomyelitis. Latterly it has been found, however, that the lesions of a localized or generalized meningitis may be present, the process being situated in the cerebrum, cerebellum, or medulla oblongata, as the case may be. In many cases (the majority of those positively recognized), the anterior horn cells are the structures most damaged. They show signs of degeneration, and as a result the motor nerve fibres arising from these cells degenerate, with resultant paralysis of the muscles supplied. Some of the motor cells in the anterior horns of the cord are wholly destroyed; others recover partially; still others recover completely. This accounts for the gradual recovery from the paralysis; for as function is regained on the part of the motor cells, regeneration of the nerve fibres takes place, and impulses that cause the muscle to contract are again transmitted.

Symptoms. The incubation period is usually about a week. In the typical case, the child is taken suddenly ill with symptoms in no wise characteristic. They are often thought to be due to tonsillitis or influenza. There is usually vomiting, sometimes diarrhea, moderate fever ranging from 101° to 103°, with the usual symptoms that accompany a febrile disturbance. There may be pain in the limbs, rigidity of the neck, and symptoms suggestive of meningitis (*q.v.*). The characteristic feature of the typical case is *paralysis,* which appears within

a week of the onset. In infants and very young children this paralysis may not be noticed for several days, though obviously present when sought. In older children, paralysis is, of course, noticed as soon as it appears. At first the paralysis may seem to affect one or more limbs in their entirety, but it soon manifests its rather characteristic distribution. The muscles are usually affected in groups (corresponding to the particular segment of the spinal cord in which the anterior horn cells have been most damaged), and are rarely affected singly. Thus, the peroneal muscles on the outer side of the leg may be involved, the extensor muscles of the front of the thigh, the deltoid group covering the shoulder, some of the muscles of the forearm, etc. Usually more than one group is involved at first. The paralysis is what is termed "flaccid," *i.e.,* the paralyzed limb lying quite loosely. There is no disturbance of sensation, though at times the limb may be cool to the touch and slightly edematous.

The actual febrile period lasts usually from a few days to two weeks. In bad cases the patient may be overwhelmed with toxemia, and die in a few days, or else death may ensue as a result of paralysis of the muscles of respiration.

After the acute febrile period has passed, the stage of repair begins, which may last as long as two years. It is important to remember that paralysis is always most extensive at first, that practically every case shows marked improvement over the condition as it existed at first, and that complete recovery occurs in about 20 per cent. of the cases.

In the course of epidemics many atypical cases are seen, some showing damage to the various cranial

nerves, as shown by facial and ocular paralyses of vary-
ing degrees of severity, others giving symptoms of a
multiple neuritis (pain along the course of certain
nerves, sensitiveness to touch and paralysis), still others
showing symptoms almost characteristic of meningitis
(*q.v.*). In the course of epidemics stress must be laid
upon the *abortive forms* of poliomyelitis, where no
paralysis occurs, although the general symptoms of ill-
ness may be present, *i.e.*, fever, together with symptoms
suggesting a respiratory, meningeal, or general influenzal
infection. Every transition stage is noticed between
these non-paralytic forms and those showing most ex-
tensive loss of function. It is now believed that in large
epidemics of poliomyelitis from one-third to one-half of
all the infections are abortive forms.

Prognosis. The death-rate varies between 10 and 40
per cent., according to statistics of various epidemics.
Death usually occurs on the fourth or fifth day, and the
mortality is greater in adults than in children.

Prophylaxis. At present, prophylaxis does not seem
to be of much service. The modes of transmission of
the virus of the disease are insufficiently known, and in
epidemics the great number of undiagnosed and abor-
tive cases as well as the numbers of healthy adults act-
ing as carriers make any attempt at satisfactory isola-
tion and quarantine practically impossible. The patient
should, of course, be isolated, and other children in the
family kept away from school for at least eight weeks.
All discharges from the nose, throat, bladder, and rec-
tum should, when possible, be destroyed by burning.
Those individuals not affected should have the nose and
mouth sprayed with some mildly antiseptic solution. In
times of epidemics schools should be closed, children's

parties not held, and all children should be watched carefully to see that they never use any article belonging to any other child. Disinfection of the sick-room is imperative, and, if possible, fumigation of the entire premises after a case of poliomyelitis is desirable.

Treatment. During the acute stage of the disease the management is that of any febrile affection. Bed, liquid diet, attention to the bowels and kidneys, cold or heat applied to painful areas, and sedatives when indicated. The management of the stage of repair in poliomyelitis is a very complicated matter, and cannot be taken up in detail. Much depends upon the competence of the nurse, and upon the zeal with which she executes the physician's orders as to the different measures to be carried out. These orders should be given in great detail, preferably in writing, as should they be misunderstood and wrongly carried out, irremediable harm may result.

The general management of the stage of repair in poliomyelitis, consists in the prolonged and judicious application of the following therapeutic agencies:

1. Electricity.
2. Massage.
3. Local heat.
4. Exercise.
5. Orthopædic apparatus (braces).
6. Surgical operations planned to help the damaged muscles in the resumption of their proper function.

CHAPTER XXVII.

EPIDEMIC CEREBROSPINAL MENINGITIS.

THIS disease occurs both in epidemics and in single scattered cases. It is most prevalent in the winter and spring months, occurs most frequently in crowded quarters where there is faulty hygiene, and may be communicated by the secretions of the nose, mouth, and conjunctiva. Whether it can be communicated by insects has as yet not been determined.

Etiology. The disease is caused by a specific germ —the *diplococcus intracellularis meningitidis of Weichselbaum.*

This organism is always present in the bodies of the leukocytes in the spinal fluid in epidemic meningitis, and has also been found in the secretions of the nose, mouth, and eye.

Pathology. The characteristic changes found are limited to the meninges of the brain and spinal cord.

1. *Brain.* The meninges (membranes) are congested and inflamed. There is an exudate of serum and pus at the edges of the brain, and extending over the upper and outer surface (the "convexity" of the brain). The brain-tissue itself is the seat of congestion, hemorrhages, thrombi, and small abscesses.

2. *Cord.* The meninges and body of the cord are affected in a manner similar to those of the brain. The central canal of the cord and the ventricles of the brain are dilated, the amount of spinal fluid is increased, the

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fluid itself is turbid, and is under much greater pressure than normal.

Symptoms. These vary greatly in various epidemics. In a typical attack, the onset is sudden, with severe headache, vomiting, and temperature from 102° to 104°. The headache increases in intensity, and becomes agonizing. There is sensitiveness to light, and sometimes to sound, the neck becomes stiff, and the head is retracted. Any attempt to bend the head forward causes excruciating pain. The reflexes (knee-jerk, wrist-jerk, etc.) are markedly increased. Kernig's sign is present (inability to extend the leg fully when the thigh is placed at right angles to the trunk).

The pupils are at first contracted, and usually equal. There may be an eruption of herpes on the lips, or else herpes zoster (shingles) may appear on the body. All the signs hitherto enumerated are those of *cerebral irritation*. With the further progress of the disease, the signs of *cerebral depression* set in. In a fully developed case the patient is in a semi-conscious, delirious, stuporous, or comatose condition, usually lying on the side in a crouching position, the head retracted, the legs drawn up, the arms bent at the elbows—*i.e.,* nature seeking the position of greatest relaxation. The high fever may fall to subnormal; the patient may be quiet, or else toss restlessly about. When aroused, he may show signs of irritability and excessive sensitiveness. There is usually an increase in the leukocytes.

The course of the disease is very varied. Excessively severe cases may result in death within a few hours; other cases may last many weeks, the fever running an irregular course. Usually the disease is at its height

for five or six days, after which the symptoms gradually abate.

The complications of cerebrospinal meningitis are not numerous, but are very serious.

1. Otitis. Common, and often resulting in deafness, which is absolute and incurable. If the patient is very young, deaf-mutism is the result.

2. Pneumonia. Frequent, and very fatal.

3. Hydrocephalus. Not so frequent, but very fatal.

Prognosis. The outlook is always very grave. Previous to the discovery of antimeningitis serum the mortality was excessively high—80 per cent. or more. Since this serum has been used, mortality has been greatly reduced, but still remains about 25 per cent.

Lumbar Puncture. Lumbar puncture is used as a method of diagnosis and as a method of treatment. It is a procedure that the nurse will never be called upon to carry out, but it is one that she will witness, and in the performance of which she will lend assistance. Therefore, she should understand what is being attempted, and what information is being sought from the procedure.

By lumbar puncture is meant the insertion of a hollow needle into the spinal canal, and the withdrawal through the needle of the spinal fluid for the purposes of :

(1) Examination.

(2) Lessening intracranial pressure.

(3) Both of the preceding.

(4) As a preliminary measure to the injection of certain drugs or sera.

For the performance of lumbar puncture the nurse in attendance should have the following articles in readiness.

(1) Needles—sterilized.
(2) Iodine.
(3) Gauze and sponges.
(4) Gloves for the physician.
(5) Sterile cotton.
(6) Sterile test-tubes (2) to receive fluid.
(7) Collodion.

The patient is turned on the side, with his back toward the operator, and drawn as near as possible to the edge of the bed. The thighs are flexed on the trunk, and the legs on the thighs, and the back is "bowed" forward as much as possible, so as to increase the space between the vertebræ. The nurse usually holds the patient in this position while lumbar puncture is being performed. Usually, no anesthetic is necessary, and the procedure is, as a rule, simple and rapid for one accustomed to it.

The site chosen for lumbar puncture is between the second and third, or third and fourth, lumbar vertebræ.

Normal spinal fluid is absolutely clear, and escapes from the needle at the rate of about 8 drops to the minute. If the fluid is under pressure, it may run in a steady stream, or spurt several feet.

The following statements are true in a general way:

If fluid is clear and under no excessive pressure, normal; if fluid is clear and under excessive pressure, tuberculous meningitis; if fluid is turbid and under excessive pressure, probably epidemic cerebrospinal meningitis or some septic meningitis; if fluid is blood-stained, no immediate inference can be drawn

Treatment. Prophylaxis. Should be the same as for typhoid fever (*q.v.*).

Thanks chiefly to the work of Dr. Simon Flexner, of the Rockefeller Institute in New York, a serum has been manufactured that greatly detracts from the terrors of

epidemic cerebrospinal meningitis. This is antimeningitis serum.

Antimeningitis serum is obtained, as is diphtheria antitoxin, from horses which have gradually been immunized to large doses of the poison of the germ causing cerebrospinal meningitis. *The serum* is of value in this disease alone, and *is of no use in any other form of meningitis.*

A lumbar puncture is done, and if the diplococci of cerebrospinal meningitis are found in the spinal fluid, the of the spinal cord. More than 30 c.c. of the pain, and in giving stimulants when needed. serum are never given at one dose. Injections are given daily for four days, and the signal for cessation of treatment is failure to find any more diplococci in the cells of the spinal fluid.

General Treatment. The remainder of the treatment of this disease is purely symptomatic, and consists in giving the patient as much nutritious food as possible, in keeping the bowels well open, in giving sedatives for pain, and in giving stimulants when needed.

CHAPTER XXVIII.

SYPHILIS.*

SYPHILIS is a specific infectious disease caused by the presence in the tissues of the infected individual of the *Spirochæta pallida.*

A nurse is practically never called upon to care for a case of syphilis as such, for those cases do not require nursing. She will, however, be called upon to nurse many individuals who have or have had syphilis, and she should realize and appreciate the enormously important rôle played by this disease in predisposing to or actually bringing about other pathological conditions. A very brief review of syphilis itself will be given, and then a few words will be said concerning its causal relationship to other diseases.

It has been determined beyond a doubt that syphilis was first brought to Europe from Española or Haiti in 1493 by the sailors with Columbus on his first voyage of discovery. The disease began to be noticed during the invasion of Italy by Charles VIII. of France in 1494, in order to conquer Naples, with an army of mercenaries from all parts of Western Europe. With the defeat of Charles's army the disease was traced by the scattering of his troops, and appeared in France, Germany, and Switzerland in 1495, in Holland and Greece in 1496, in England and Scotland in 1497, and in Hungary and Russia in 1499.

As is usual with the initial appearance of a disease among a people unused to its presence, during the first

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* For the subject matter of this chapter I am mainly indebted to the very interesting monograph, "Syphilis as a Modern Problem," by Dr. W. A. Pusey, of Chicago.　　　(233)

decades of its prevalence syphilis raged with extraordinary severity; but at the end of fifty years Europeans had developed a certain amount of immunity to it; and the cases becoming milder and more chronic, assumed the type seen today.

Syphilis is usually acquired during sexual intercourse, and the first sign of it, the "initial lesion" or chancre, is therefore most frequently to be found on the genitals. Infection cannot take place unless the mucous membrane is broken, but the abrasion may be of microscopic size. Individuals in the so-called "secondary" stage of the disease, with "mucous patches" in their mouths, can infect others by kissing, the initial lesion then showing itself on the lips or in the mouth. Shamberg reports a case where a young man with a chancre of the lip infected 7 young girls at a party where kissing games were played.

The course of syphilis falls naturally into several stages. A brief summary of these stages may serve to give a clear picture of the disease.

1. *Incubation Period*. From the time of infection to the appearance of the initial lesion. Approximately four weeks.

2. *Primary Stage*. Lasting about six weeks. During this period the chancre, which is a small ulcer, with a hard base, and covered by a small amount of clear secretion, develops and disappears; the infection gradually invades the entire body, and the period is abruptly brought to an end by the appearance of the eruption or rash.

3. *Secondary Stage*. Begins with the appearance of the rash, and ends only with the disappearance of evidences of an active systemic infection. It may last

for a few weeks, several months, or more than a year, and may be accompanied by slight fever, some loss of weight, and a mild degree of general malaise. The rash may assume one of many forms.

4. *Tertiary Stage.* Characterized by the presence of lesions due to isolated local syphilitic processes, situated anywhere in the body, and affecting most frequently the blood-vessels, liver, brain, central nervous system, and bones.

Syphilis thus presents resemblances to three types of disease. First, a local infection characterized by a local lesion, the chancre; second, it resembles the acute specific infections, especially the eruptive fevers; third, by the formation of localized foci of inflammation, it resembles tuberculosis. The secondary manifestations of syphilis are those of an acute systemic disease. The tertiary manifestations are those of a chronic disease sharply localized in its activity.

Syphilis rarely directly kills the patient. Its most dreaded effects are remote, and are exerted upon various organs and tissues of the body.

A few historical facts of very recent date may be of interest as showing the epoch-making contributions of the twentieth century to the knowledge of this important disease.

In 1903 Metchnikoff and Roux demonstrated that apes could be inoculated with syphilis.

In 1905 Schaudinn and Hoffman discovered the spirochæta pallida, which is the sole cause of syphilis.

In 1906-7 Wassermann, Neisser, and Bruck developed the "Wassermann reaction" as a test for the presence and diagnosis of syphilis, which has enabled many thousands of cases to be recognized and treated that could not have

been diagnosed by any of the previous methods at the physician's command.

In 1909-10 Ehrlich discovered and gave to the profession "606," or "salvarsan," a preparation of arsenic for the treatment of syphilis.

Hereditary Syphilis. Syphilis can be, and often is, directly transmitted from the mother to the child she is bearing. If the child is born actively syphilitic, it usually dies in a few weeks at most, and during its life it is acutely ill. The skin and mucous membranes are markedly affected by the syphilitic eruption; there is severe running at the nose, and laryngitis; the nasal discharge is purulent, and the child snuffles and breathes with difficulty. Because of the laryngitis it frequently has a characteristic high-pitched harsh cry. It is emaciated, and the liver and spleen are usually enlarged.

If the child is born infected, but with no active symptoms, these begin in from two to six weeks. Snuffles is usually the first symptom, to be followed by those described in the preceding paragraph. If treated, many of these cases recover.

For a woman to bear a syphilitic child, she must be herself actively syphilitic. If the disease is inactive a syphilitic mother will bear healthy children, while later, if the disease reawakens, she will bear syphilitic children.

Prophylaxis of Syphilis. This cannot be dealt with here. The question is a vast one, indeed, and is as much sociological as medical. Syphilis and its spread are so intimately connected with the questions of prostitution, loose morals, the relations of the sexes, that to touch the question at all would be to plunge into very deep water.

Treatment of Syphilis For several centuries mercury has been known as a specific remedy for the syph-

ilitic poison. It has been administered by inunction, by mouth, and hypodermically. It is still one of the main-stays of treatment.

Salvarsan is the newest substance for combating syph-ilis. Ehrlich hoped that by the administration of salvar-san intravenously he could definitely cure syphilis. This hope has not been fully realized. Valuable as salvarsan is, it has been found that the best results are obtained when it is combined with vigorous treatment with mercury.

Syphilis in Its Relationship to Other Diseases.

This aspect of syphilis is one of the most important to be considered. Diseases directly or remotely due to the syphilitic poison may appear many years after all symp-toms and signs of syphilis have disappeared, and, up to the present time, no method of treatment has been de-vised that will prevent the later effects of this disease.

By the action of the syphilitic poison on the blood-ves-sels, arteriosclerosis is frequent, and there are authori-ties who assert that every case of aneurysm not due to injury, has, as at least one of its causative factors, syph-ilis. As a result of arteriosclerosis the kidneys are dam-aged, and the heart overtaxed, giving rise to the condi-tion described as cardiovascular-renal disease.

It has been shown beyond a doubt that locomotor ataxia and paresis (softening of the brain) are always late results of syphilis. These two diseases, both very serious and incapable of cure (arrest being the best re-sult attainable by any form of treatment), form one of the saddest chapters occurring in middle life as a remote effect of a syphilitic infection received perhaps a quar-ter of a century before.

By its action in lowering bodily resistance, syphilis plays a part in the causation of almost every disease known to man.

Sir William Osler has said that "the man that knows syphilis in all its manifestations, knows most of medicine"; and it is important for the nurse to appreciate the rôle played in the human body by this disease. One of the reasons that syphilis has so many remote effects is that the disease in its active form is singularly yielding to treatment. Symptoms disappear rapidly, and apparent health returns. The patient realizes with difficulty that from one to three years of treatment are necessary to thoroughly eradicate the spirochæta pallida. With a return of physical well-being treatment is maintained half-heartedly, if indeed it is not wholly abandoned, and many of the spirochætas, unharmed, bury themselves deep in the tissues, where they hibernate in safety, to make their activities felt in after years in the form of a variety of pathological conditions.

CHAPTER XXIX.

LOCOMOTOR ATAXIA.
(TABES DORSALIS.)

LOCOMOTOR ataxia is a disease of the sensory portion of the central nervous system, and is characterized anatomically by a sclerosis or fibrous-tissue formation in the posterior columns of the spinal cord, known as the columns of Goll and Burdach. It has been established that this disease is always a late result of syphilis, and may occur in an individual that has to all appearances been cured of that infection, and in whom no symptoms whatsoever have been present for as much as twenty-five years.

Symptoms.

1. *Incipient stage:*
    (1) Pain. Sharp, stabbing, called "lightning pains"; most common in the legs.
    (2) Ocular symptoms. Paralysis of external muscles of the eye, ptosis (drooping of upper lid), and Argyll Robertson pupil, in which the pupil loses its sensitiveness to light, but continues to react to accommodation.
    (3) Difficulty in voiding urine.
    (4) Loss of patellar reflex (knee-jerk).

Any or all of these symptoms may exist for several years, the patient remaining in a stationary condition.

2. *Ataxic stage.* This develops gradually. "One of the first indications to the patient is inability to get about readily in the dark, or to maintain his equilibrium

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when washing his face with the eyes shut. When the patient stands with the feet together and the eyes closed, he sways and has difficulty in maintaining his position, and he may be quite unable to stand on one leg. He does not start off promptly at the word of command. On turning quickly he is apt to fall. He descends stairs with more difficulty than he ascends them.

"Gradually the characteristic ataxic gait develops. The patient, as a rule, walks with a stick. The eyes are directed to the ground, the body is thrown forward, and the legs are wide apart. In walking the leg is thrown out violently, the foot is raised too high, and is brought down in a stamping manner, with the heel first, or the whole sole comes in contact with the ground.

"Ultimately the patient may be unable to walk without the assistance of two canes. This gait is very characteristic, and unlike that seen in any other disease. The incoordination is not only in walking, but in the performance of other movements. It may early be noticed by a difficulty which the patient experiences when buttoning his collar, or when performing one of the ordinary routine acts of dressing. One of the most striking features of the disease is that with marked inco-ordination there is no loss of muscular power" (Osler). Shifting pains persist, and render many patients miserable. Attacks of severe pain referable to various organs of the body may occur. The so-called gastric crises are the most important—attacks of pain in the stomach, accompanied by nausea and vomiting.

3. *Paralytic Stage*. After the ataxic stage has persisted for an indefinite time, the patient gradually loses the power of walking, and becomes bed-ridden or paralyzed. In this stage the condition known as "surgical

kidney" (or ascending infection of the urinary tract) is apt to occur, or the patient may succumb to some infection such as pneumonia or tuberculosis.

Prognosis. Recovery is impossible, for certain fibres in the spinal cord are permanently destroyed. Arrest at any stage is often possible.

Treatment. Antisyphilitic treatment energetically instituted is indicated in practically every case. Salvarsan combined with mercury gives the best results. Large doses of potassium iodide, formerly extensively used, are no longer considered advisable. The patient must be placed under the best possible hygienic surroundings, well fed, the avenues of elimination kept open, and particular attention paid to the skin, the nutrition of which is often interfered with. If bed-sores develop in these patients, they are apt to run a rapid and virulent course, and may prove fatal.

Fränkel has devised some special exercises for the re-education of co-ordination which are of value. The general treatment of locomotor ataxia, apart from the antisyphilitic medication and Fränkel's exercises, is largely symptomatic, and in a disease characterized by so many symptoms, and extending over such a lengthy period of time, a recital of all methods employed would be too voluminous.

CHAPTER XXX.

DIABETES MELLITUS.

DIABETES is a disease of nutrition, in which sugar (glucose) cannot be utilized in the usual way; hence it appears in the blood, and is excreted in the urine (glycosuria), the amount of which is greatly increased.

Etiology. Heredity seems to play an important part. The disease is most frequent after 40 years of age, and, generally speaking, the earlier in life an individual is affected, the more severe is the type of the disease, and *vice versa*. Diabetes often develops after an infection or an injury.

The pathology is cloaked in such complexity and obscurity that no mention will be made of it.

Symptoms. The disease is gradual and insidious in its onset. Often the first symptom is sugar in the urine, which is accidentally discovered in the course of a routine examination, as, for instance, for life insurance. Other symptoms which are rather characteristic are:

1. Excessive hunger.

2. Excessive thirst.

3. The passing of an excessively large amount of pale straw-colored urine—from 3 to 10 quarts in twenty-four hours.

4. Emaciation and increasing weakness.

5. Crops of boils or carbuncles.

6. Intense itching, often about the genitals.

7. The characteristic urinary findings:

    (*a*) Large amount of urine voided.

    (*b*) High specific gravity—1.030 to 1.045.

    (*c*) The presence of sugar.

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In untreated cases the disease progresses steadily, and a condition known as acidosis appears—*i.e.,* the over-loading of the body with acids. Substances known as beta oxybutyric acid and acetone appear in the urine. The appearance of these chemical compounds pave the way for the last act in the evolution of the disease.

8. Diabetic coma. This may come on with weakness, a sweetish odor to the breath due to acetone, somnolence, and gradually developing unconsciousness, the patient dying in a few hours. Diabetic coma may begin with nausea, vomiting, headache, delirium, great distress, and dyspnea. Finally there are cases in which without any previous warning the patient is seized with headache, a sense of intoxication, and rapidly passes into a deep coma which ends in death.

Complications. The manifestations in the skin have been mentioned. It is a fact that diabetics show very little resistance to infection, and that in them trivial wounds and scratches become the starting-point for a spreading cellulitis, ending often in gangrene. Pulmonary tuberculosis is not infrequent, and is very fatal. Albuminuria is also of fairly common occurrence.

Prognosis. In untreated cases of diabetes the outlook is absolutely bad. Under careful management much can be done for the patient, though it is not possible to speak of "cure" in connection with this disease. However, in the mild and moderately severe types, judicious treatment will prolong life for years under very comfortable conditions, but vigilance must never be relaxed, for "the roots of sin are there." and if the patient insists on exceeding his dietetic limitations he soon pays for his indiscretion by a return of the old train of symptoms. In the severest types of the disease but little can be done.

Treatment. The treatment of diabetes is receiving a great deal of attention at present, and is in a "transition" stage, for many ideas that had taken root firmly are threatened in their security by the work of Dr. Frederick M. Allen and his so-called "starvation treatment." The problem of the management of the diabetic is becoming better understood, and the outlook for the patient is becoming proportionately more hopeful.

The first thing to be done with a diabetic is, of course, to determine his "carbohydrate tolerance"—in other words, to discover how much carbohydrate food he can care for without sugar appearing in the urine. In order to determine this factor, the urine must be first rendered sugar-free. This is done by one of two methods:

    (1) Gradual withdrawal of carbohydrate foods.
    (2) Sudden and complete withdrawal of all foods
        (Allen's "starvation method").

1. *Gradual Withdrawal of Carbohydrate Foods.* The patient is placed on an ordinary diet for a couple of days, and the amount of sugar in the twenty-four-hour urine is determined. He is then placed on a "strict" or carbohydrate-free diet (see accompanying list, compiled by Dr. E. P. Joslin, of Boston), plus 100 Gm. white bread (= 60 Gm. carbohydrate), for three days. Each day the amount of sugar in the twenty-four-hour urine is determined. In very mild cases the urine may become sugar-free in three days or less. In moderately severe cases the sugar output may fall to between 20 Gm. and 10 Gm. In severe cases the amount of sugar may equal the 60 Gm. of carbohydrate taken in, or even exceed that amount.

In mild cases and in those of medium severity, after the urine has been rendered sugar-free, 20 Gm. of white

bread may be added to the diet every other day until
sugar reappears in the urine; and when this occurs, the
total amount of white bread tolerated is easily known.
This amount must never be exceeded, though substitu-
tions can be made as shown by the accompanying con-
venient table:

"Accessory" or "Carbohydrate-containing" Diet (to be
used in prescribed amounts only).

	Carbohydrate Content Percentage	Amount in grams. = 20 Gm. White Bread. = 12 Gm. Carbohydrate.
White bread	60	20
Coarse black bread	50	22
Zwieback	70	17
Oatmeal	67	18
Rice or macaroni	70 to 80	15 to 17
Cocoa	30	40
Potato	18 to 20	60 to 70
Turnip	7 to 10	120 to 170
Jerusalem artichoke	15 to 20 (inulin)	60 to 80
Cherries	12 to 14	80 to 100
Grapes, plums, peaches, apples, pears	10	120
Melons, strawberries, raspberries, blackberries	5	240
Milk	4.8	250
Kefir	2.4	500
Cream (thick)	3	400
Beer	4 to 5	240 to 300
Oranges	6	200

Thus, if a patient were allowed, say, 80 Gm. white bread (= 48
Gm. sugar), he might, if he cared to, substitute therefor:
    (1) 44 Gm. black bread + ½ liter milk, or
    (2) 30 Gm. white bread + 150 Gm. potato, or
    (3) 30 Gm. rice + ½ liter milk, etc.
                    ("Monographic Medicine," *Barker*).

While testing carbohydrate tolerance, sodium bicarbonate should be given when the carbohydrate intake falls below 60 Gm.

2. *Sudden and Complete Withdrawal of All Foods.* This method of managing diabetes has been in use but since 1914; the results are as yet inconclusive. It holds forth such promise, however, and is so extensively in use among those best qualified to judge, that the nurse should be familiar with the general principles involved.

Under this method of treatment the patient is compelled to fast until the urine is sugar-free, and for 24 or 48 hours longer. In severe cases alcohol (being a food that does not cause glycosuria) is given. Sodium bicarbonate is also administered for the first few days, but is then discontinued. After the urine has been sugar-free for from 24 to 48 hours, feeding is undertaken very slowly and cautiously, but not according to any fixed program, since it is desirable to individualize the diet to suit the special need of the particular patient. The only requirement is that the urine shall remain sugar-free. The appearance of even a trace of sugar is the signal for a fast day. The original fast may last from two to ten days; subsequent fasts, according to Allen, need never exceed one day.

To obtain carbohydrate tolerance, 200 Gm. of vegetables of 5 per cent. class (see Joslin's table), are added, increasing until sugar appears in urine. A fast day is at once instituted, and, the carbohydrate tolerance being known, the limit is not exceeded.

Protein tolerance is also estimated by adding gradually to the dietary eggs and meat, until sugar appears in the urine, or a quantity of protein sufficient for the needs of the body is being consumed.

In some cases fat tolerance must be determined as well. During the fast, patients lose considerable weight. This Allen considers beneficial. Later the patient is permitted to gain weight as long as he can do so without the reappearance of glycosuria.

The accompanying schema and tables of Dr. Joslin (pp. 248-9) give in concrete form a working basis for the management of cases of diabetes by the Allen method.

Drugs play a very small part in the management of cases of diabetes. At one time opium in large doses (usually in the form of codeine) was widely used. Arsenic has many adherents. Many other drugs have been recommended, but have failed to be of lasting benefit.

Diabetic Coma. As coma is one of the manifestations of acidosis, the patient should be saturated with alkalies. Sodium bicarbonate is usually used, by mouth, by rectum or intravenously. Free catharsis, obtained usually by use of salines is indicated. Oxygen by inhalation may lessen the air-hunger and dyspnea. Recovery from coma is "rare but not impossible."

Itching. This is often almost intolerable. Prolonged warm soda baths are of aid, and mild solutions of ammonia water and phenol help. Vaginal douches are of aid, and scrupulous cleanliness of the parts is important.

In diabetes, as in almost no other disease, the nurse can be of inestimable aid to the physician and to the patient. With a knowledge of the principles of treatment, with reference to tables giving food values, and with ability to make the very simple test for the *presence* of sugar (*not the quantity present*), she can take a case along from day-to day very satisfactorily, and can make the small individual changes and variations in the patient's dietary, thus relieving the doctor from details.

STRICT DIET. *Meats, Fish, Broths, Gelatine, Eggs, Butter, Olive Oil, Coffee, Tea, and Cracked Cocoa.*

Foods Arranged Approximately According to % of Carbohydrates.

	5%		*10%*	*15%*	20%
Vegetables (fresh or canned)	Lettuce Cucumbers Spinach Asparagus Rhubarb Endive Marrow Sorrel Sauerkraut Beet greens Dandelion greens Swiss chard Celery	Tomatoes Brussels sprouts Water cress Sea kale Okra Cauliflower Egg plant Cabbage Radishes Leeks String beans Broccoli	Pumpkin Turnip Kohl-Rabi Squash Beets Carrots Onions Mushrooms	Green peas Artichokes Parsnips Canned lima beans	Potatoes Shell beans Baked beans Green corn Boiled rice Boiled macaroni
Fruits	Ripe olives (20% fat) Grape fruit		Lemons Oranges Cranberries Strawber's Blackberr's Gooseberr's Peaches Pineapple Watermel'n	Apples Pears Apricots Blueberries Cherries Currants Raspberries Huckleberr's	Plums Bananas Prunes
Nuts	Butternuts Pignolias		Brazil nuts Bk walnuts Hickory	Almonds Wal'ts (Eng.) Beechnuts	Peanuts
Misc.	Unsweetened and unspiced pickle; clams, oysters, scallops, liver, fish roe.		Pecans Filberts	Pistachios Pine nuts	40% Chestnuts

* Reckon available carbohydrates in vegetables of 5% group as 3%, of 10% group as 6%.

(30 grams 1 oz.) Contain approximately	Carbohydrates G.	Protein G.	Fat G.	Calories
Oatmeal, dry wgt.	20	5	2	120
Cream, 40%	1	1	12	120
Cream, 20%	1	1	6	60
Milk	1.5	1	1	20
Brazil nuts	2	5	20	210
Oysters, six	4	6	1	50
Meat (uncooked, lean)	0	6	3	50
Meat (cooked, lean)	0	8	5	75
Bacon	0	6	15	155
Egg (one)	0	6	6	75
Vegetables 5 and 10% group	1 or 2	0.5	0	6 or 10
Potato	6	1	0	25
Bread	18	3	0	90
Butter	0	0	25	225
Fish, cod, haddock (cooked)	0	5	0	20
Broth	0	0.7	0	3
Small orange or ½ grapefruit	10	0	0	40

Fasting. Fast until sugar-free. Drink water freely, and tea, coffee and clear meat broth as desired. In very severe, longstanding and complicated cases, without otherwise changing

habits or diet, omit fat; after two days omit protein and halve carbohydrate daily to 10 Gm., then fast.

Carbohydrate Tolerance. When the twenty-four-hour urine is sugar-free, add 150 Gm. of 5 per cent. vegetables, and continue to add 5 Gm. carbohydrates daily up to 20, and then 5 Gm. every other day, passing successively upward through the 5, 10, and 15 per cent. vegetables, 5 and 10 per cent. fruits, potato and oatmeal to bread, unless sugar appears or the tolerance reaches 3 Gm. carbohydrate per kilogram body weight.

Protein Tolerance. When the urine has been sugar-free for two days, add 20 Gm. protein (3 eggs) and thereafter 15 Gm. protein daily in the form of meat until the patient is receiving 1 Gm. protein per kilogram body weight, or if the carbohydrate tolerance is zero, only ¾ Gm. per kilogram body weight.

Fat Tolerance. While testing the protein tolerance, a small quantity of fat is included in the eggs and meat given. Add no more fat until the protein reaches 1 Gm. per kilogram (unless the protein tolerance is below this figure), but then add 25 Gm. daily until the patient ceases to lose weight or receives not over 40 calories per kilogram body weight.

Reappearance of Sugar. The return of sugar demands fasting for twenty-four hours or until sugar-free. The diet is then increased twice as rapidly as before, but the carbohydrate should not exceed half the former tolerance until the urine has been sugar-free for two weeks, and it should not then be increased more than 5 Gm. per week.

Weekly Fast Days. Whenever the tolerance is less than 20 Gm. carbohydrate, fasting should be practised one day in seven; when the tolerance is between 20 and 50 Gm. carbohydrate, upon the weekly fast day 5 per cent. vegetables and one-half the usual quantity of protein and fat are allowed; when the tolerance is between 50 and 100 Gm. carbohydrate, the 10 and 15 per cent. vegetables are added as well. If the tolerance is more than 100 Gm. carbohydrate, upon weekly fast days the carbohydrate should be halved.

1 gram protein,	4 calories	1 kilogram = 2.2 pounds.
1 " carbohydrate,	4 "	30 grams (g) or cubic centimeters
1 " fat,	9 "	(c.c.) = 1 ounce.
1 " alcohol,	7 "	A patient "at rest" requires
6.25 " protein contain 1 g. nitrogen		25 calories per kilogram body weight.

CHAPTER XXXI.

THE BLOOD.

THE blood is the nutritive medium of the body, distributes food and oxygen to all the tissues, and takes from them waste products to be delivered to the organs of elimination. It comprises about one-thirteenth of the body weight.

The blood is composed of a liquid portion, blood-plasma, in which are floating the cellular elements or corpuscles of the blood:

1. Red blood-cells—erythrocytes.
2. White blood-cells—leukocytes.
3. Blood-platelets.

Plasma. Normally, when free from corpuscles, plasma in thin layers is clear and colorless. When seen in thicker layers it has a faint yellow tinge. Blood upon escaping from the blood-vessels usually clots. The clot in its formation shrinks and squeezes out a fluid having a slightly yellow tinge. This is known as blood-serum. It may be said that plasma is the liquid part of blood *before* clotting, and serum the liquid part of the blood *after* clotting. In the blood-serum are contained many of the substances that figure so largely in the different phases of immunity. These will be touched on in the chapter on "Immunity."

Clotting. One of the most remarkable properties of blood is its power to coagulate or clot immediately or very shortly after escaping from the vessel. This power of clotting saves the life of each one of us countless times, for were it not for the blood-clot, we would all

die of hemorrhage from the most trivial injury. There are indeed individuals known as hæmophiliacs, or bleeders, whose blood clots very slowly, if at all, and who frequently bleed to death from some very trifling cut or operation.

"The essential part of the blood-clot is the fibrin. Fibrin is an insoluble proteid which is absent from normal blood. In blood that has been shed—and, under certain conditions, in blood while still in the blood-vessels—this fibrin is precipitated, if the word may be used, in the form of an exceedingly fine network of delicate threads, which permeates the whole mass of blood, and gives the clot its jelly-like character. The shrinking of the threads causes the subsequent contraction of the clot.

"If the blood has not been shaken in the act of clotting, almost all the red corpuscles are caught in the fine fibrin meshwork, and as the clot shrinks these corpuscles are held more firmly, only the clear liquid of the blood being squeezed out, so that it is possible to get specimens of serum containing few or no red corpuscles. The leukocytes, on the contrary, although they are also caught at first in the forming meshwork of fibrin, may readily pass out into the serum in the later stages of clotting, on account of their power of ameboid movement (see chapter on Immunity). If the blood has been agitated during the process of clotting, the delicate network will be broken in places, and the serum will be more or less bloody—that is, it will contain numerous red corpuscles.

"If during the time of clotting the blood is vigorously whipped with a bundle of fine rods, all the fibrin will be deposited as a stringy mass upon the whip, and the remaining liquid part will consist of serum plus the bloodcorpuscles. Blood which has been whipped in this way

is known as 'defibrinated' blood. It resembles normal blood in appearance, but is different in its composition: it cannot clot again" (An American Textbook of Physiology).

The cellular elements of the blood.

1. *Red Cells or Erythrocytes.* These are small bi-concave disks, practically round when normal, and having in the fresh state a yellowish color when looked at under the microscope. They are very numerous, there being in men about 5,000,000 to the cubic millimeter, and in women about 4,500,000. A normal red cell is never nucleated.

In anemia from any cause, the red cells are reduced in number and changed in character—to what extent depends upon the severity of the anemia. The lowest red blood-count on record is 143,000 per cubic millimeter.

The following changes may take place in red cells as a result of severe anemia:

(*a*) Great pallor, due to deficient amount of hemoglobin.

(*b*) Poikilocytosis, or irregularity in outline.

(*c*) Nucleation (normoblasts), due to the throwing into the circulation by the bone-marrow of young immature forms, in order to supply the crying need for blood-cells.

(*d*) Appearance of large nucleated red cells (megaloblasts), representing still more immature forms thrown out when the body's need for new cells is most urgent.

(*e*) "Stippling" of red cells—a form of degeneration.

2. *White Cells or Leukocytes.* These cells are far less numerous than the red cells, a normal leukocyte count showing from 4000 to 7000 per cubic millimeter. There

are several varieties of leukocytes. The following table
gives the main varieties and the approximate percentage
in normal blood:

	Per cent.
Polymorphonuclear neutrophile	65
Small lymphocyte	20
Large lymphocyte	10
Eosinophile	3
Basophile (mast-cell)	2
	100

The leukocytes, as mentioned above, are capable of
motion by means of their power of ameboid movement,
and are very active as scavengers of the body, and as
taking a prominent part in the fight against infection.
This function is referred to in the chapter on Immunity.

Leukocytosis. By leukocytosis is meant an increase
in the number of leukocytes in the blood. Practically all
leukocytosis is pathological—*i.e.,* is called forth by the
presence of an enemy in the form of some infection.
The exception to this rule lies in those blood diseases
known as the leukemias, where, owing to an abnormality
of the blood-forming organs, especially the spleen, a vast
number of immature leukocytes are flung into the cir-
culation. Leukocytosis occurs in all infections and in-
fectious diseases except:

1. Typhoid fever.
2. Uncomplicated tuberculosis.
3. Malaria.
4. Influenza.
5. Measles.
6. Mumps.
7. Leprosy.

The usual count when a moderate leukocytosis exists
is from 15,000 to 30,000. Occasionally the count will be

as high as 50,000 or 75,000. In the leukemias the white cells may number 500,000 to the cubic millimeter, and even more.

The presence or absence of a leukocytosis is often of great value in diagnosis, and the nurse should appreciate its importance and learn to understand its significance in conditions where the count is frequently made. The following rules may prove of aid:

1. If the infection is severe, and the patient's resistance good, leukocytosis is early, marked, and persistent.

2. If infection and resistance are both less marked, but fairly well proportioned one to the other, leukocytosis still occurs, but comes later, is less in degree, and ceases more quickly.

3. If the infection is one of unusual virulence, as in the so-called "fulminating" cases of sepsis, diphtheria, or pneumonia, *no leukocytosis occurs.*

4. Occasionally when the infection is unusually mild, and the resistance unusually good, there may be little or no leukocytosis.

Hemoglobin. The hemoglobin is the coloring matter of the red cells, and is the substance to which the blood owes its red color. A chemical change in the hemoglobin in combination with each red cell is responsible for the fact that arterial blood is bright red, and venous blood a deep crimson. (See chapter on the Circulation.)

Hemoglobin estimations are very frequently made, the test being the most simple of any applied to the blood. Normal hemoglobin content ranges from 90 per cent. to 110 per cent. on the scale with which the blood under examination is compared, and in all probability treat-

ment would rarely be instituted with a normal red cell count and hemoglobin 80 per cent. or over.

Hemoglobin varies pathologically in three ways:

1. Proportionately to the loss in red cells—*i.e.*, with a red cell count of 3,750,000 (25 per cent. less than normal) a hemoglobin reading of approximately 75 per cent.

2. Relatively high as compared with the number of red cells. This condition occurs in all anemias of the pernicious type—*e.g.*, a red cell count of 2,000,000 (a loss of almost 60 per cent. of red cells), and a hemoglobin reading of approximately 55 per cent. (In order to be in proportion to the red cell loss, the hemoglobin reading should be in the neighborhood of 40 per cent.)

3. Actually low and relatively low as compared to the number of red cells. This relationship occurs particularly in chlorosis—that anemia of young girls that seems to consist almost entirely in a hemoglobin deficiency— *e.g.*, red cell count 4,000,000, hemoglobin 35 per cent.

In addition to estimating the percentage of hemoglobin, and the number of red and white cells, a differential white cell count is often done in order to determine whether there is any change in the percentage of the various types of leukocytes, as such changes are often of aid in diagnosis. No attempt will be made to dwell upon results obtained from differential white cell counts, as the nurse is in no wise concerned with them.

CHAPTER XXXII.

PERNICIOUS ANEMIA AND LEUKEMIA.

PERNICIOUS anemia is "a chronic and usually fatal disease of unknown origin, producing, especially in elderly men, paroxysms of intense anemia, and usually degeneration of the spinal cord" (Cabot).

The cause of pernicious anemia is as yet unknown. Many factors have been suggested, and some authorities believe it to be the result of an infection resembling in a way syphilis. This theory is based upon the following factors:

1. Fever occurring in pernicious anemia.

2. Enlargement of the spleen.

3. Frequency with which the spinal cord is attacked.

4. Beneficial effects of arsenic, and especially of salvarsan, given in a manner similar to that employed when treating syphilis.

The pathology of the disease is complicated and obscure, and will not be dealt with. The disease is not uncommon, affects mainly individuals between 40 and 60 years of age, and is found in men twice as frequently as in women.

Symptoms. A characteristic of pernicious anemia is its insidious onset. *General weakness* is complained of in every case. The early cases are extremely difficult to recognize, but when the condition is moderately advanced the patient's color is very suggestive. A yellowish pallor is present, the patient having a dead, waxy tinge, dif-

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ferent from that seen in other forms of anemia. It is a color that must be seen to be appreciated; no amount of description can do it full justice. In connection with general weakness the patient also complains of other symptoms, some of which are characteristic of any severe anemia, others of which point more or less directly to the pernicious type.

Those symptoms present in any severe anemia are: dyspnea, palpitation, headache, vertigo, and less frequently, edema.

Those symptoms more or less characteristic of pernicious anemia are:

1. Gastro-intestinal attacks or "crises"—paroxysms of severe abdominal pain, practically uninfluenced by treatment, passing off at the end of a variable length of time, and often followed by a period of improvement.

2. Diarrhea. Continuous or paroxysmal.

3. Symptoms suggestive of tabes dorsalis (locomotor ataxia) (*q.v.*).

Usually there is but slight loss of weight as compared with the general weakness. The most characteristic symptoms of pernicious anemia are to be found in

The blood:

The total quantity of blood is lessened.

Blood-pressure is extremely low.

On pricking the finger the drop of blood may look quite red, but its *watery* condition is at once apparent. The red cells are usually found to be below 2,000,000 to the cubic millimeter (normal being from 4,500,000 to 5,000,000). The hemoglobin may be 50 per cent. of normal, while the red cells may be but 40 or 30 per cent. of normal. This condition is characteristic of pernicious anemia, and is the result of nature's effort, in view of the

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very great destruction of red cells, to supply each remaining cell with as great a percentage of hemoglobin as possible. In addition, it may merely be mentioned in passing that the outlines of the red cells are irregular instead of being smooth and round, that the average size of the red cells is increased, due to young, immature forms being cast into the circulation to make up for the loss in cells, and that red cells containing nuclei are found.

The course of the disease is characterized by periods of marked improvement followed by periods of increase in all symptoms. The blood-picture varies with the general symptoms, sometimes improving to a remarkable degree, only to grow worse again. The outlook for permanent recovery is bad, but if the patient reacts satisfactorily to treatment, life may be maintained for several years. The blood-picture rarely, if ever, reaches normal, but periods of improvement may last from three months to two years.

Treatment. Rest in bed for a time, fresh air, food in abundance are, of course, indicated. The general management is very similar to that employed in pulmonary tuberculosis (*q.v.*). Arsenic is the one drug that seems to exert a favorable effect on the course of the disease. Most physicians have their favorite preparations and mode of administration. The three following preparations have found the most favor:

1. Fowler's solution by mouth.
2. Cacodylate of sodium hypodermically.
3. Salvarsan or neosalvarsan intravenously.

Attention should also be directed toward the intestinal tract with a view to keeping it as clean as possible by means of colon lavage and intestinal antiseptics.

Leukemia. This is a disease of one of the blood-forming organs, especially the bone-marrow. Nothing is known as to its causation. The condition is rare.

There are two main classes:

    1. Myeloid.

    2. Lymphoid.

1. *Myeloid. Symptoms:*

    (1) Enlarged spleen. Usually reaches the navel; may extent into the pelvis.

    (2) Dyspnea.

    (3) Intestinal disturbances, due to pressure from enlarged spleen, and to dragging of the spleen on its ligaments.

    (4) General loss of strength.

    (5) Blood-picture. Leukocytes enormously increased in numbers. Usually 300,000 to 1,-500,000 per cubic millimeter (normal 4000 to 7000). Abnormal forms known as myelocytes present in large numbers. Moderate anemia.

2. *Lymphoid. Symptoms:*

    May be acute and begin with weakness, fever and hemorrhages from various portions of the body. May be glandular enlargement.

    Dyspnea. Spleen enlarged—less so than in myeloid form, but almost always present.

    Blood-picture. Leukocytes markedly increased, averaging about 180,000 per cubic millimeter and consisting practically entirely of lymphocytes.

Course. These two forms of leukemia are generally chronic from the start, and usually end fatally, though,

as in the case of pernicious anemia, there are frequently extended periods of improvement.

Treatment. X-ray treatment at the hands of an experienced operator has given the best results. No drug exerts any appreciable effect on the course of the malady.

CHAPTER XXXIII.

EXOPHTHALMIC GOITRE
(Graves' Disease.)

Exophthalmic goitre is a disturbance of nutrition due to a disordered condition of the thyroid gland. This gland, like the adrenal glands, the ovaries, etc., furnishes to the body what is known as an "internal secretion"— *i.e.,* a secretion that is not given off through a duct, but that comes off from the body of the thyroid itself and spreads about through the tissues. The nature of this secretion is not well understood, but its presence is essential to life. It is generally believed that exophthalmic goitre is due to an excessive secretion on the part of the thyroid gland, the evidence for this belief resting mainly on two facts:

1. The conditions of myxedema and cretinism, which are positively known to be due to *insufficient* thyroid secretion, present a picture which is diametrically opposite to that found in exophthalmic goitre.

2. Cases of exophthalmic goitre are almost invariably made worse by the administration of thyroid extract, which contains the active principle of the secretion of that gland.

Exophthalmic goitre is not a rare condition, and is assuredly met with, especially in its milder forms, more frequently than is generally believed to be the case. The earlier forms are so like mild cases of neurasthenia that this diagnosis is more frequently made.

Women are more frequently affected than men in the proportion of 8:1. The disease is one of early and

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middle adult life, occurring usually between the ages of 16 and 40. As predisposing causes are mentioned emotional shocks and worry, though it is probable that in these cases the disease was latent, and that its symptoms began to show themselves after bodily, and especially nervous, resistance had become lowered. The actual cause of the disease is unknown.

Symptoms. There are five characteristic symptoms of exophthalmic goitre:

1. Goitre.
2. Exophthalmos—bulging of the eyeballs.
3. Tachycardia—rapid heart action.
4. Tremor.
5. Nervousness.

The onset of the disease is very gradual, the patient usually complaining for some weeks or months of increasing nervousness, palpitation, shortness of breath, and inability to perform ordinary duties without undue fatigue.

In a well-developed typical case the neck is prominent from the swelling of the thyroid gland, the eyes are staring and bulge perceptibly, the heart action is rapid— from 120 to 150 per minute—there is palpitation accompanied often by a choking sensation, and slight exertion brings on marked shortness of breath. When the hands are held out and the fingers spread apart as far as possible a very fine tremor is observed in them, and as a rule the hands sweat profusely. There is intense nervousness; the patient starts at the slightest sound. There is inability to concentrate the attention on anything for any length of time; and the patient's spirits are poor. The appetite is bad, the tongue is coated, and constipation is frequent. There is almost always marked loss of

weight and insomnia. At times there is a low grade of
fever, but this is not a prominent symptom, save in the
most severe cases.

Some of the classical symptoms of exophthalmic
goitre are very often absent, notably that of goitre. The
protrusion of the eyes may be extremely slight, but
cases are on record in which it has been so marked that
the eyes could not be closed, and eventually the eyeballs
sloughed away.

Tachycardia, tremor, nervousness, with slightly staring
eyes are the symptoms most commonly noted.

Prognosis. It has been said that exophthalmic goitre
is "a disease from which patients never recover and
never die." This is hardly true—it is better to say that
"few recover and some die." Recovery in the fullest
sense of the word is not frequent, the best results usu-
ally being the restoration of the patient to a condition
which enables her to lead a happy and useful life, but one
during which she must be ever careful not to overdo, and
during which she must take longer or shorter periods of
rest in order to tide over the times when the thyroid
again begins secreting too actively.

Treatment. As far as the general management is
concerned, a routine is indicated which strongly re-
sembles that advocated for cases of early pulmonary
tuberculosis (for details see chapter on Tuberculosis),
consisting in rest in the open air and abundant, nutri-
tious, and easily digestible food. Nervousness is gen-
erally best dealt with by means of continued rest, warm
baths at night, and the administration of full doses of the
bromides when necessary. It is not considered good
practice to give morphia or any of the preparations of
opium to these patients.

Iodine is a drug that sometimes helps these patients, and sometimes seems to make the condition worse. When used it is usually given in the form of potassium iodide and the syrup of the iodide of iron. Iron in some form is often given, as there is usually a moderate degree of anemia.

The use of extracts of the thymus gland, and the use of thyroidectin, which is a substance made from the blood of sheep whose thyroid glands have been removed, have benefited some cases, while in the majority of instances they have failed.

Surgical Treatment. Surgery has been of greater benefit to cases of exophthalmic goitre than has medical treatment. Two main surgical procedures are in use:

1. Resection of a portion of the gland (usually not over two-thirds).

2. Ligation of two or three of the four thyroid arteries in the hope of lessening the activity of the gland by limiting its blood-supply.

Probably the best treatment for a case of exophthalmic goitre is surgery in the hands of an expert, together with careful previous and subsequent general management on the part of the general practitioner.

CHAPTER XXXIV

IMMUNITY.

IMMUNITY is defined as "exemption from disease," or that condition of the body which enables it to resist infection. Immunity and infection, though opposites, are so intertwined that mention cannot be made of one without reference to the other.

Immunity can be classified as follows:

1. Natural immunity. Thus, the human race is naturally immune to chicken cholera, and animals are naturally immune to measles.

2. Acquired immunity. Thus, one attack of typhoid fever usually renders an individual immune to that disease.

A certain amount of immunity may be acquired toward a particular disease by its frequent occurrence. When syphilis first appeared among Europeans, its ravages were frightful; but within fifty years, as a result of the countless number of cases that appeared, individuals developed a relative immunity to the disease, and as a result its manifestations were not as terrible.

When an individual is attacked by an infection, the defensive resources, or immunizing forces of the body, are called upon to do their part in repelling the invader. The stronghold of the defensive resources of the body is the blood. Certain cells of the blood play an active part in resisting infection, and many substances present in the blood-serum play their part in safeguarding the organism.

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Phagocytosis. By this is meant the power of the white blood-cells to ingest, kill, and digest bacteria. Elie Metchnikoff, of Paris, is the man to whom science owes an unpayable debt for his labor in demonstrating the act and consequences of phagocytosis. It is known that the leukocytes are capable of motion by projecting a portion of their body in the shape of a long finger-like process. The rest of the body of the leukocyte is then drawn up to the finger-like projection, and thus the leukocyte moves. This is known as "ameboid" movement. When an infection exists, leukocytes at once come to the battle-ground, and a struggle ensues between them on the one hand, and the bacteria on the other. The leukocytes by their power of ameboid movement surround one or more bacteria, engulf them, and digest them. This can be observed under the microscope, the bacteria being seen to be engulfed, to grow less and less distinct within the body of the leukocyte, and finally to become wholly invisible. If the infection is not of excessive virulence, and if the leukocytes are healthy and plentiful, the body wins the fight against the bacteria and recovery ensues. This is why it is always a good sign in an acute infection, such as lobar pneumonia or appendicitis, to have the blood show a high leukocyte count. If, however, the infection is very virulent, and the body defenses inadequate, no leukocytosis results and death ensues.

Metchnikoff has shown that acquired immunity is largely due to stimulated phagocytosis. This is true whether the immunity is due to one attack of the disease or to vaccination. A rabbit which has been artificially immunized to anthrax shows a more marked phagocytosis on inoculation with a virulent culture than does a rabbit that has not been artificially immunized.

In the blood-serum are found many substances that play a rôle in the production of immunity. Among these may be mentioned:

1. Precipitins.

2. Agglutinins. Substances that cause bacteria to clump, and upon whose presence is based the Widal reaction, so valuable in the diagnosis of typhoid fever.

3. Opsonins. Substances that, as it were, prepare and make ready bacteria, so that they can more readily be engulfed by the leukocytes in the process of phagocytosis.

The theories of the mechanism of immunity are extremely complex, and no attempt will be made to describe them here. Paul Ehrlich, of Berlin, has elaborated the chief among them, his famous "side chain" theory of immunity, which in brief is as follows: A cell possesses normally certain defensive forces or receptors which will unite with a certain amount of toxin and neutralize it, thus protecting the cell. When a cell is threatened with attack by a toxin, it is stimulated to the production of other receptors, or "side chains," and immunity comes about when there is such an overproduction of these side chains that there are more than enough to neutralize every bit of toxin that is attacking.

Acquired immunity may be:

1. Active.

2. Passive.

In specific treatment of various infections it is sought in some cases to produce an active immunity, in others a passive immunity.

By an active immunity is meant that the body, stimulated by the infecting bacteria, or by the injection of those same bacteria killed (*i.e.,* a vaccine), manufactures its own resisting forces, brings up its own reserves, and

actively fights its own battle. Vaccination against typhoid fever is a good example of the production of an active immunity, for, as a result of the injection of a certain number of killed typhoid bacilli, the body is stimulated to such an overproduction of receptors for typhoid toxin that it can resist infection with live typhoid bacilli, and not become ill with the disease.

By passive immunity is meant that the body is supplied from outside with its means of defense, ready to use, and requiring no effort at all on the part of the individual.

The best example of the production of a passive immunity is to be found in the antitoxin treatment of diphtheria. Here, a certain amount of antitoxin secured from another artificially immunized animal (the horse) is injected, and at once is able to neutralize the diphtheria toxin circulating in the patient's blood. This neutralization goes on with no effort whatsoever on the part of the patient, whose rôle is merely passive. This form of immunity can be used with success only in those diseases that are purely toxic, *i.e.*, in which the infecting bacteria themselves do practically no harm, but only the poisons liberated by those bacteria. Hence, the use of antitoxic sera, while giving brilliant results in purely toxic diseases such as diphtheria and tetanus, have unfortunately but a very limited range of application.

To quote Vaughan: "Now we have the great problem of infection and immunity fairly before us. It is a contest between bacteria and body-cells, and .   .   .   they are armed with similar weapons. The bacterial cells have their enzymes, poisons, and toxins. The body-cells have their enzymes, bactericidal, and bacteriolytic agents, opsonins and phagocytes. The phagocytes constitute the

mobile army of defense, and the fixed cells elaborate destructive weapons. Which of these bears the brunt of the defense depends upon the armament of the invader."

If the invasion is mainly bacterial in its nature, the leukocytes are called upon to play the principal part in winning the victory. If the invasion is mainly toxic, the tissue-cells have to bear the brunt of the defense. If the invasion is both bacterial and toxic, all the "arms of the service" play an equally important part in saving the body from destruction.

GLOSSARY.

Aëration. The state or process of being supplied with air or gas.

Agglutinin. A specific principle occurring in the blood-serum of an animal affected with a disease of microbic origin, and capable of causing the clumping of the bacteria peculiar to that disease.

Albuminuria. The presence of albumin in the urine.

Alveolus. An air-cell of the lung.

Ambulant. Referring to a patient that is up and about. Not confined to bed.

Anemia. Deficiency of blood as a whole, or deficiency of the number of red corpuscles or of the hemoglobin.

Anasarca. An accumulation of serum in the subcutaneous areolar tissues of the body.

Anesthetic. Any drug that causes insensibility to pain.

Anorexia. Loss of appetite.

Antitoxin. A counterpoison or antidote manufactured by the body to counteract the toxins of bacteria.

Anuria. Absence of secretion of urine.

Aorta. The main arterial trunk of the body arising from the left ventricle of the heart.

Aphasia. Partial or complete loss of the power of expressing ideas by means of speech or writing.

Aphonia. Loss of the voice.

Apoplexy. Hemorrhage from a blood-vessel in the brain.

Arteriosclerosis. A chronic inflammation of the arterial walls resulting in more or less extensive fibrous tissue formation.

Ascites. Fluid in the peritoneal cavity.

Auricle. One of the two upper and smaller chambers of the heart.

Bactericidal. Having the power of killing bacteria.

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Bacteriolytic. Possessing a disintegrating action upon living bacteria.

Basophile. A leukocyte whose granules stain with basic dyes.

Bronchitis. Inflammation or catarrh of the bronchial tubes.

Buttock. The fleshy part of the body back of the hip-joint formed by the masses of the glutei muscles.

Calorie. The amount of heat required to raise a kilogram of water, one degree Centigrade.

Capillary. A minute blood-vessel connecting the smallest branches of the arteries with those of the veins.

Carbohydrate. An organic substance containing 6 carbon atoms, or some multiple of 6, and hydrogen and oxygen in the proportion in which they form water (H_2O)—that is, twice as many hydrogen as oxygen atoms. ($C_6H_{10}O_5$.)

Carbon dioxid. A gas chemically known as CO_2 or carbonic acid gas.

Cardiac. Relating or pertaining to the heart.

Catharsis. Purgation.

Chlorosis. A form of anemia most common in young women, characterized by a marked reduction of hemoglobin in the blood, with but slight diminution of red corpuscles.

Chordæ tendineæ. The tendinous strings connecting the papillary muscles of the heart with the mitral and tricuspid valves.

Chorea. A functional nervous disorder usually occurring in youth, characterized by irregular and involuntary action of the muscles of the extremities, face, etc., with general muscular weakness. (Syn., St. Vitus' Dance.)

Cicatrization. Scar formation.

Clinical. Relating to bedside treatment.

Coma. Unconsciousness from which the patient cannot be aroused by external stimulus.

Compensation. The extra work performed by a leaking heart in order to maintain the balance of the circulation.

Crisis. The sudden termination of a fever.

Cusp. A flap of a heart-valve.

Cyanosis. A bluish discoloration of the skin from deficient oxydation of the blood.

Cyanotic. Referring to an individual exhibiting cyanosis.

Cystitis. Inflammation of the urinary bladder.

Delirium. A condition of mental excitement with confusion and usually hallucinations and delusions.

Deoxygenated. Deprived of oxygen.

Desquamation. A shedding of the superficial epithelium of the skin. "Peeling."

Detritus. Unrecognizable or formless waste matter.

Diaphoresis. Sweating.

Diaphragm. The muscular and tendinous plane separating the thorax from the abdomen.

Diastole. The period of rest in the cardiac cycle.

Dicrotic. The term applied to a pulse-beat in which with every wave the examining finger feels a double beat.

Diffusible. Spreading to all parts of the body.

Diplococcus. A coccus or round germ occurring in pairs.

Diuretic. Any drug that increases the flow of urine.

Dysphagia. Painful swallowing.

Dyspnea. Shortness of breath.

Edema. Presence of serum in the subcutaneous tissues.

Embolus. A particle of fibrin or other material brought by the blood-current and forming an obstruction at its place of lodgment.

Empyema. Pus in the pleural cavity.

Endocarditis. Inflammation of the endocardium.

Endocardium. The lining membrane of the heart.

Engorgement. Congestion.

Enzyme. A digestive ferment.

Eosinophile. A leukocyte whose granules stain with acid dyes.

Epidemic. A term applied to a disease affecting a large number or spreading over a wide area.

Epiglottis. A cartilaginous structure situated behind the root of the tongue that prevents food and drink from passing into the larynx.

Epithelium. A term applied to the group of cells that covers the skin and that lines all canals having communication with the external air, as the mouth, urethra, intestine, etc., and that are specialized for secretion in certain glands, as the liver, kidney, etc.

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Erosion. Eating away.

Eruption. A rash.

Erythrocyte. A red blood-corpuscle.

Exophthalmic. Relating to exophthalmos.

Exophthalmos. Protrusion of the eyeballs.

Exudate. The material that has passed through the walls of blood-vessels into adjacent tissues.

Febrile. Having fever.

Feces. The movements of the bowels.

Fibrin. A proteid found in blood after it has been shed, and constituting the main factor in the clotting of blood.

Fibrosis. Formation of fibrous tissue.

Flagellated. Bearing hair-like processes or flagellæ.

Focus. The location of an infection.

Fomites. Any substance that absorbs and transmits a contagion.

Gastric. Relating or pertaining to the stomach.

General anasarca. Serum in the tissues, and in the peritoneal and pleural cavities.

Germicide. Any substance having the power of killing germs.

Glomerulus. One of the secreting elements in the kidneys, lying in the cortex of that organ, and formed of a tuft of capillaries surrounded by a capsule (Bowman's), and giving off a uriniferous tubule.

Goitre. A swelling of the thyroid gland not of inflammatory origin.

Gout. A disease of metabolism characterized by attacks of pain in the small joints, and by a deposit therein of sodium urate.

Hemophiliac. An individual in whose blood the power of clotting is reduced or absent—a "bleeder."

Hemoglobin. The coloring matter of the blood. Found in the red blood-corpuscles.

Hemorrhagic. Pertaining to hemorrhage. Bloody.

Hepatization. The name applied to the second and third stages in the consolidation of the lung in lobar pneumonia.

Herpes. An acute inflammatory condition of the skin characterized by the development of a group of vesicles.

Herpes zoster. An eruption occurring along the course of the intercostal nerves. (Syn., "Shingles.")

Hydrocephalus. A collection of fluid within the ventricles of the brain, or outside the brain, between it and the skull.

Hydrotherapy. Treatment by means of water.

Hydrothorax. Fluid in the pleural cavity.

Hyperemia. Congestion.

Hyperplasia. Excessive formation of tissue. An increase in the size of a tissue or organ owing to an increase in the number of cells.

Hypertrophy. An increase in the size of a tissue or organ independent of the general growth of the body.

Hyperpyrexia. Excessively high fever—over 106° F.

Hypertension. Blood-pressure that is above normal.

Hypnotic. Any drug that produces sleep.

Hypochondrium. The upper lateral region of the abdomen beneath the lower ribs.

Hypodermoclysis. The subcutaneous injection of fluid.

Hypotension. Blood-pressure that is below normal.

Incontinence. Lack of control over the contents of either bladder or rectum.

Incubation. The period of a disease between the onset of the infection and the development of symptoms.

Infection. (1) The communication of disease from one body to another. (2) The agent that produces disease.

Infectious. Having the power of communicating disease.

Infusion. The intravenous injection of salt solution.

Ileum. The third portion of the small intestine.

Immunity. The condition of the body in which it resists the development of disease.

Inhibitory. Checking. Restraining.

Inoculation. The act of introducing the virus of a disease into the body.

Insomnia. Inability to sleep.

Intercostal. Relating to any structure situated between the ribs.

Interstitial. Pertaining to interstitial or connective tissue.

Intracranial. Situated within the skull.
Invasion. The onset of a disease.

Jejunum. The second portion of the small intestine.

Koplik's spots. Small bluish spots seen in cases of measles, and occurring on the mucous membrane of the cheeks and lips *before* the appearance of the rash. An absolutely diagnostic sign of measles

Laryngitis. Inflammation of the larynx.
Leukocyte. A white blood-corpuscle.
Leukocytosis. An increase in the number of leukocytes.
Ligation. The tying of a blood-vessel.
Lumen. The cavity surrounded by the walls of a tubular vessel.
Lymphocyte. A variety of leukocyte having a very large nucleus and a relatively small cell-body.
Lysis. The gradual disappearance of a fever.

Macule. A spot upon the skin not elevated above the surrounding level.
Malaise. A general feeling of illness, accompanied by restlessness and discomfort.
Media. The middle coat of the wall of an artery.
Megaloblast. A large nucleated red blood-corpuscle.
Meninges. The dura mater, pia mater, and arachnoid membranes of the brain and spinal cord.
Metabolism. The group of phenomena whereby organic beings transform foodstuffs into complex tissue-elements, and convert complex substances into simple ones in the production of heat and energy.
Metastatic. Referring to metastasis, which is the transfer of a diseased process from one part of the body to another by means of the blood or lymph channels.
Meteorism. Gas in the intestines.
Motile. Possessing the power of motion.
Mucoid. Resembling mucus.
Myocarditis. Inflammation of the heart-muscle.
Myxedema. A disease of nutrition due to lack of secretion or absence of the thyroid gland.

Necrosis. Death of tissue.

Necrotic. Referring to necrosis.

Nephritis. Inflammation of the kidneys.

Neutrophile. A leukocyte whose granules stain with neutral dyes.

Normoblast. A red blood-corpuscle of normal size having a nucleus.

Nucleated. Possessing a nucleus.

Nucleus. The essential part of a typical cell.

Oliguria. A deficient amount of urine.

Opsonin. A substance in blood-serum that prepares bacteria for digestion by leukocytes.

Organic. Pertaining to the animal and vegetable world.

Otitis. Inflammation of the ear.

Oxydation. The act or process of combining with oxygen.

Oxygen. A colorless, tasteless, odorless gas, composing one-fifth of atmospheric air, and in the absence of which human and animal life cannot be maintained.

Palpitation. Consciousness of the heart-beat.

Papule. A small circumscribed solid elevation of the skin.

Paracentesis. Puncture of the wall of one of the cavities of the body, *e.g.*, the ear, the pleura, the peritoneum.

Parasite. An animal or vegetable living upon or within another individual, the host, *e.g.*, malarial parasite, in red blood-corpuscles; tape-worm, in intestines.

Parenchymatous. Referring to the parenchyma or specialized portion of an organ, as differentiated from the supporting and surrounding tissue.

Pathogenic. Producing disease.

Pathological. Referring to pathology, which is that branch of medical science that treats of the modifications of function and changes in structure caused by disease.

Pericarditis. Inflammation of the pericardium.

Pericardium. The membrane covering the heart and the root of the aorta and pulmonary artery.

Peritoneum. The membrane lining the interior of the abdominal cavity and surrounding the contained viscera or organs.

Peritonitis. Inflammation of the peritoneum.

Phagocytosis. The process of ingestion and digestion of micro-organisms by the leukocytes.

Phlebitis. Inflammation of a vein.

Physiology. The science of the normal workings of the human body.

Pitting. The formation of a pit or hollow by pressure upon edematous subcutaneous tissue.

Pleura. The membrane surrounding the lung.

Pleurisy. Inflammation of the pleura.

Pneumococcus. The causative factor in lobar pneumonia.

Pneumonia. Inflammation of the lung.

Pneumothorax. Air in the pleural cavity.

Poikilocytosis. Irregularity in outline of red blood-corpuscles.

Polymorphonuclear. A leukocyte having nuclei of varied shapes and sizes.

Polyuria. An excessive amount of urine.

Precipitin. A substance present in blood-serum capable of producing a precipitate in a clear solution of the particular albumin or culture filtrate against which the individual whose blood is used, has been immunized.

Prophylactic. Preventive.

Proteid. Any of the important and essential nitrogenous constituents of animal and vegetable tissues.

Ptosis. A drooping or sagging. May refer to drooping of the eyelid or to a general sagging down of the abdominal viscera.

Purulent. Containing pus.

Pus. A liquid substance consisting of cells and an albuminous fluid formed in certain kinds of inflammation.

Pyemia. A disease due to the presence in the blood of pus-forming germs.

Pyelonephritis. Inflammation of the kidney and its pelvis.

Receptor. One of the so-called "side arms" of a cell, which according to Ehrlich's "side chain" theory of immunity is for protection of the cell by uniting with an attacking molecule of toxin.

Regurgitation. The back-flow of blood through a heart valve that is defective.

Remission. A fall in fever in which, however, the temperature still remains above normal.

Resection. The process of cutting out and removing.

Resolution. The fourth stage in the evolution of the process undergone by the inflamed lung in lobar pneumonia.

Sedative. A drug whose action is to quiet the patient.

Semilunar. The valves guarding the openings of the aorta and pulmonary artery.

Sepsis. Blood-poisoning.

Septicemia. Blood-poisoning.

Serous. Relating to serum.

Serum. The clear yellow fluid that separates itself from the clot after the shedding of blood.

Sordes. The crusts that accumulate on the teeth and lips in continued fevers.

Splanchnic. Pertaining to or supplying the viscera.

Sputum. The secretion of the lungs and bronchi.

Stenosis. Constriction of a heart valve, as a result of which it cannot open as fully as it should.

Stippling. A term used to describe a peculiar spotted appearance of red blood-corpuscles in severe anemias.

Striated. Possessing striæ or stripe-like lines.

Stupe. A cloth used for applying heat or counterirritation.

Stupor. A state of partial unconsciousness from which the victim can be roused.

Systole. The period of contraction or "work-period" of the heart.

Tachycardia. Excessively rapid heart-action.

Thrombosis. The formation of a thrombus.

Thrombus. A clot of blood formed within the heart or blood-vessels.

Toxemia. Blood-poisoning. A condition in which the blood contains poisonous products, either those produced by the body-cells, or those due to the growth of micro-organisms.

Toxin. A poison.

Tracheotomy. The operation of opening the trachea or windpipe and inserting a tube through which the patient can breathe.

Tremor. A trembling of the voluntary muscles.

Tubercle. The specific lesion produced by the tubercle bacillus.

Vagus. The 10th pair of cranial nerves.

Vascular. Pertaining or relating to the circulatory system.

Vasodilator. A drug causing dilatation of the arteries and consequent lowering of blood-pressure.

Venesection. The operation of opening a vein in order to allow the escape of a certain amount of blood. "Bleeding."

Ventricle. (1) One of the two lower larger chambers of the heart. (2) One of several spaces in the brain.

Virulent. Having the nature of a poison.

Vomitus. The material that is vomited.